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A TEXT-BOOK

OF

PRACTICAL MEDICINE,

WITH PARTICULAR REFERENCE TO

PHYSIOLOGY AND PATHOLOGICAL ANATOMY.

BY

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DISEASES OF THE URINARY ORGANS.

SECTION I.

DISEASES OF THE KIDNEY.

CHAPTER I.

HYPERÆMIA OF THE KIDNEY.

Hyperæmia of the kidney and its consequences are not to be confounded with inflammation of the kidney, although during life it is sometimes quite impossible to distinguish between the two, and notwithstanding that similar symptoms appear in both disorders, such as the presence of blood and albumen in the urine, as well as the appearance in it of peculiar objects, generally called fibrinous casts (although it is probable that they are not composed of fibrin, and certainly are not pure fibrin, but consist in great part of mucin).

Etiology.—A thorough knowledge of the normal circulation of the kidney is indispensable to a proper understanding, not only of the physiology, pathogeny, and etiology, but also of the symptoms of hyperæmia of this organ. To *Virchow* the credit is due of having corrected some of our former ideas upon this subject, and of having rendered others more complete.

According to the result of his researches, the branches of one particular portion of the renal artery (that, namely, which belongs to the middle and outer part of the cortical substance) go exclusively to form the afferent vessels, and, entering the Malpighian capsules as such, these divide to form the vessels of the glomeruli. Then, leaving the Malpighian capsule as efferent vessels, they again break up into branches, once more to reunite as renal veins. On the boundary between the cortical and pyramidal substances there is a sort of "neutral ground." Here there are arteries from which branches arise, some forming afferent vessels, glomeruli, and efferent vessels, with long off-

shoots running into the cortical and medullary substance, and others which act directly as nutrient vessels for the medullary substance. Finally, the renal artery has branches upon which there are no glomeruli at all, and whose function is simply nutrition of the medullary substance.

The resistance encountered by the blood which passes through the glomeruli is far greater than that met by the blood which merely flows directly from the arteries into the capillaries. Even under normal conditions the blood-pressure in the renal arteries is a very considerable one, that vessel being both short and of disproportionately large calibre. When the pressure within these arteries is increased, hyperæmia of course will first arise in that portion of the kidney where the blood encounters the greatest resistance, namely, in the cortical substance, and, above all, in the glomeruli. Where the resistance is less, as in the medullary substance, although the circulation is accelerated there, the actual amount of blood which the part contains is not materially increased. *It is very different, however, when the escape of the blood from the renal veins is impeded.* In such a case the quantity of blood in the veins and capillaries is augmented, but the engorgement cannot extend through the narrow efferent vessels into the glomeruli; and, as the contents of the arteries are abnormally reduced in most of the disorders in which there is an obstruction of the renal veins (as in cardiac and pulmonary diseases), the reason at once becomes apparent why, even in cases of extreme obstructive hyperæmia of the kidney, the glomeruli are scantily supplied with blood, and the secretion of urine is proportionately small. Perhaps, too, nervous influence may not be without effect in bringing about this condition, since it is possible that in the kidneys, just as in other organs, the arteries of the various vascular systems are not subject to the same kind of innervation.

The causes which induce fluxion to the kidney are:

1. The transient plethora induced by every copious draught of liquid. This hyperæmia is most pronounced in the secretory portion of the kidney, and the profuse transudation which takes place out of the overloaded glomeruli is the principal step in the process by which the general plethora of the system is relieved.

2. There is a second cause of renal hyperæmia, which is closely analogous to the first, and which occurs when the left side of the heart is hypertrophied, and which also is confined to the arterial system, including the glomeruli.

3. Collateral fluxion to the kidney may result from compression of the abdominal aorta, or iliac arteries, by a tumor or gravid uterus, as well as from derangement of the circulation in the capillaries of the

skin in the cold stage of intermittent fever. In the second stage of Bright's disease, compression of the vessels of the cortical substance, by the distended urinary tubules, also gives rise to collateral fluxion in the medullary substance of the kidney (*Virchow*).

4. Dilatation of the afferent vessels, from palsy of their muscular elements, seems to be the principal cause of that arterial hyperæmia of the kidney, the existence of which is revealed by the discharge of a large quantity of limpid urine in certain spasmodic diseases (urina spastica).

5. Fluxionary hyperæmia is found in the vicinity of inflamed regions and deposits of morbid product. This form of hyperæmia we have already endeavored to account for, in treating of an analogous condition in other organs, ascribing it to a loss of tone in the tissues, and to a dilatation of their capillaries, whose walls are ill supported by the surrounding relaxed parts.

6. Renal hyperæmia complicates inflammatory disease of the urinary passages, especially disease of the pelvis of the kidney.

7. The renal hyperæmia which sometimes attends the use of cantharides, balsam of copaiba, and similar drugs, as well as that which accompanies certain infectious diseases, especially scarlatina, measles, typhus, and cholera, seems to be of similar origin. However, the affections of the kidney which arise from the diseases above named cannot all be attributed to hyperæmia. Many of them belong to the class of maladies which we shall describe as parenchymatous degeneration of the kidney, in Chapter VII., and which, undoubtedly, are capable of developing without the occurrence of hyperæmia. This is equally true of the renal affections which so often accompany pregnancy.

Obstructive engorgement of the kidney arises from causes similar to those which induce engorgement of the liver (see Volume I.), so that the two diseases often accompany one another; but it will readily be understood, from what has already been said as to the peculiar character of the hepatic circulation, that engorgement of the liver usually is of earlier occurrence and greater intensity than engorgement in the kidneys. Engorgement of the kidney arises: 1. In uncompensated valvular disease of the heart; 2. In structural disease of the heart, which depresses the functional energy of the organ; 3. When the vigor of the heart is impaired by a condition of marasmus; 4. In disease of the lungs, which causes compression or wasting of the pulmonary capillaries; 5. In conditions in which the aspiration of the blood to the thorax is arrested; 6. In rare instances, contraction and closure of the vena cava or emulgent veins, by compression or thrombosis, give rise to engorgement of the kidney, but the hyperæmia arising in such cases is of very great intensity.

Anatomical Appearances.—A kidney in a state of recent hyperæmia is of a more or less dark-red color. Sometimes it is enlarged, owing either to dilatation of its vessels or to serous infiltration. The œdema of its parenchyma and subcapsular connective tissue renders the hyperæmic organ unnaturally moist and soft, and loosens its capsule. When cut into, and when the vessels of the glomeruli are much distended by blood, the Malpighian coils dot the surface of the section, as dark-red points.

When the hyperæmia is of long standing, particularly when there is habitual engorgement, as occurs in chronic disease of the heart and lung, the changes which arise are of a different kind. The kidney is then but little enlarged, or remains of its normal magnitude, or may even be somewhat smaller than natural. It is more resistent in texture, and is of a uniform red color. Upon microscopic examination, the epithelium of the urinary tubules of the cortical substance is found to be swollen, the contour of the cells is ill-defined, and they are filled with finely-granular contents, which clear up upon the addition of acetic acid. Sometimes there is a desquamation of the degenerated epithelium here and there; and the tubules collapse after the epithelium is expelled, causing the surface of the kidney to become uneven by depressions of varying depth. *Traube* and *Beckman* have called particular attention to the difference between this condition and the degenerative chronic inflammation of the kidney, which we shall describe in Chapter IV. as chronic Bright's disease.

While the alterations above described are going on in the epithelium of the cortical substance, the straight tubules of the medullary substance are usually found filled with a material which is sometimes transparent and pale, and sometimes of a more yellow color. By moderate pressure upon the pyramids, a large quantity of an opaque creamy liquid is discharged from the papillæ, which contains a great deal of epithelium and a few of those casts of the tubuli, in the form of homogeneous transparent tolerably firm cylinders.

Symptoms and Course.—The kidney has so few nerves of sensation, and its capsule is so distensible, that hyperæmic swelling of the organ is never accompanied by pain. As the amount of urine secreted depends chiefly upon the degree of pressure within the vessels of the glomeruli, its secretion necessarily becomes more profuse in that form of renal hyperæmia which involves the arterial system of the kidney, including the vessels of the Malpighian tufts. This is almost the only symptom caused by fluxion to the kidney, which arises from copious drinking, hypertrophy of the left heart, compression of the aorta or iliac arteries, and dilatation of the renal arteries. The urine is copious in quantity, dilute in quality, of low specific gravity, and of a pale

color. In such cases, the pressure in the glomeruli hardly ever is sufficient to occasion transudation of albumen, or to rupture the vessels, and cause extravasation of blood into the Malpighian capsules. This fact is in accordance with the results of physiological experimentation. Ligation of the abdominal aorta below the origin of the renal arteries, in spite of the increase of pressure which ensues in those arteries, never results in albuminuria.

A very different train of symptoms follows upon a moderate degree of obstructive engorgement of the kidney. Since, as we have just shown, in almost every case of obstruction of the renal veins the degree of tension within its arteries is very small, there is a diminution instead of an increase of the secretion of urine. On the other hand, the strain upon the capillaries becomes exceedingly severe, since they cannot discharge their contents into the already overloaded veins until the pressure within them exceeds that within the veins. Hence, not only does the plasma of the blood readily escape from the capillaries into the urinary tubules, so that the scanty, concentrated, dark-colored urine contains albumen, and the so-called fibrinous casts (or, more properly speaking, exudation casts), but the delicate walls of the capillaries give way before the strain, so that the urine is also full of blood-corpuscles. According to the recent observations of *Liebermeister*, the simultaneous appearance of blood and albumen is so usual in obstructive hyperæmia of the kidney, that the appearance of albumen alone, without a trace of blood, almost excludes the idea of simple engorgement from the diagnosis, and indicates the existence of inflammation. This fact in pathology, which may be verified in almost every case of chronic disease of the heart, and traced through all its phases, agrees with the results of physiological experiment. Albuminuria and hæmaturia are the never-failing consequence of ligation of the renal veins, or of the vena cava above their point of entrance. The escape of plasma from the capillaries of the kidneys, in obstructive congestion, into the uriniferous tubules is analogous to the extravasation from the pulmonary capillaries into the air-vesicles, which occurs in engorgement of the lungs, and which is there called hypostasis. The so-called hypostatic pneumonia is quite as independent of genuine inflammatory action as is the disorder of the kidney at present under consideration.

The form of renal hyperæmia which apparently proceeds from relaxation of the tissues of the kidney and consequent dilatation of its capillaries, has no effect either in augmenting or diminishing the quantity of urine secreted. It is attended, however, by a more or less profuse transudation of blood-plasma, as well as by a greater degree of shedding, and probably too by a more active reproduction of the cel-

lular contents of the uriniferous tubules. Hence, when, after the abuse of irritating diuretics, or in disease which we know to be frequently complicated with this form of renal hyperæmia, we find the urine to be albuminous and full of casts thickly studded with epithelial cells, our diagnosis may be renal catarrh. This name, which has been applied to the form of hyperæmia in question by most modern pathologists, is not quite appropriate, it is true, as the tubules have no mucous membrane, and as the term catarrh itself means an affection peculiar to such a membrane. However, it is quite as appropriate as is the term catarrhal pneumonia. (See Vol. I.)

The course of both fluxionary and obstructive hyperæmia of the kidney, when the exciting cause is of a transient character, usually is favorable, and the disease in itself probably never causes death. Although, during the last few weeks of a case of heart-disease, this affection appears in its most intense form, yet it is not renal hyperæmia of which the patient finally dies, but the respiratory derangement, the dropsy, and other symptoms which proceed immediately from the cardiac disorder. However, it cannot be denied that the albuminuria aggravates the dropsy and hydræmia, and aids in undermining the strength of the patient. The renal catarrh also runs a favorable course as a rule, and, when the primary disease tends toward recovery, usually terminates in complete restoration to health. Far more rarely, diffuse parenchymatous inflammation of the kidney may develop from renal catarrh.

Treatment.—The measures called for in treatment of the cause of hyperæmia of the kidney may be inferred from the account above given of the causes themselves. When the hyperæmia of the kidney is but a symptom of a more wide-spread and grave disease, it is to the latter rather than to the hyperæmia to which the treatment should be directed Where it has arisen from the abuse of irritating diuretics, their employment must be discontinued, and all application of vesicants, and use of irritating salves upon suppurating surfaces (a very common cause of fluxion to the kidney), must be abstained from. Besides this, large quantities of drink must be administered, in order as much as possible to dilute the acrid matter which has been secreted in the kidneys. Pure water or the dilute acids are the most suitable for the purpose. The old practice of using barley-water, linseed-tea, milk of almonds, and the like mucilaginous or oleaginous liquids, must be regarded as obsolete, as it is well known that such articles have no effect upon the character of the urine.

General and local blood-letting, and derivatives to the skin or intestines, are only to be resorted to in fulfilling the indications from the disease itself, when such remedies are indicated for other reasons, or

when the hyperæmia is extreme, and there are no counter-indications against blood-letting.

CHAPTER II.

HÆMORRHAGE FROM THE KIDNEY.

ETIOLOGY.—The causes of hæmorrhage from the kidney are: 1. Wounds, contusions, and other injuries of the organ. The presence of calculi in the pelvis of the kidney is one of the most common sources of such injury. *Rayer* tells about a patient with renal calculus who had hæmaturia whenever he was compelled to ride. 2. Hæmorrhage from the kidney may proceed from intense hyperæmia, and rupture of the overloaded renal capillaries, and especially from that form of hyperæmia which accompanies the first stage of inflammation of the kidney, and which ensues after the employment of irritating diuretics, as well as in that form of the affection which follows scarlatina, small-pox, typhus, malarial fevers, and other infectious diseases. The hyperæmia which exists about the seat of a parasite, or a neoplastic growth, especially cancer, often gives rise to hæmaturia. Finally, the excessive obstructive engorgement of the kidney, produced by disease of the heart and lungs, often results in an escape of blood from the renal capillaries. 3. In rare instances renal hæmorrhage depends upon a *hæmorrhagic diathesis*, that unknown disorder of the blood-vessels from which scorbutus and purpura hæmorrhagica proceed. 4. In the Isle of France, Brazil, and some other tropical regions, hæmorrhage from the kidney is endemic, there being no known cause for the disease.

A peculiar form of renal hæmorrhage, hæmorrhagic infarction, arises under conditions similar to those in which hæmorrhagic infarction takes place in the spleen. This can be traced in most cases to embolism.

Genuine renal apoplexy is usually the result of severe injury, but in children may also proceed from severe hyperæmia (*Rokitansky*).

ANATOMICAL APPEARANCES.—In hæmorrhage from the kidney, the blood may be effused into the normal interstices of the tissues without detriment to the latter. In this way ecchymoses arise, in which there are spots of varying size stained of a deep red, and from which blood flows freely when they are cut into. They are situated sometimes under the albuginea, or within the tissues of the kidneys themselves.

In variola and diseases of that class, besides ecchymoses in the renal tissue, the mucous membrane lining the pelvis of the kidney is usually of an even, dark-red color, decidedly thickened (hæmorrhagic infiltration), and rough on the surface.

When recent, hæmorrhagic infarction has the appearance of a dark-red deposit of a cuneiform shape, the point of the wedge being directed toward the hilus of the kidney. When of longer standing, the deposit

loses its color, beginning at the middle, and becomes converted into a yellow, caseous mass, or else breaks down, forming a renal abscess, with yellow puruloid contents, which at first consist of detritus alone, but afterward are mixed with pus. Finally, these caseous or purulent masses also are reabsorbed, and at the site of the former infarction there is left a cicatricial contraction.

The seat of renal apoplexy is generally the medullary portion of the kidney. Collections, of varying size, form in the lacerated parenchyma, their contents consisting partly of clotted blood, and in part of crushed and broken-down *débris* of the tubules. It would seem that the contents, both of apoplectic extravasations and of hæmorrhagic infarctions, may undergo fatty degeneration and absorption, and that recovery may take place, leaving a depressed cicatrix. Part of the blood effused into the tubules coagulates, forming cylinders, which are densely studded with blood-corpuscles. Sometimes we find pigment in the tubules and Malpighian capsules, as the residue of some former extravasation of blood.

Symptoms and Course.—The occurrence of renal hæmorrhage does not become recognizable unless the blood be effused into the tubules, and discharged with the urine. Hence it not unfrequently happens, in *post-mortem* examinations, that hæmorrhagic infarctions and apoplectic extravasations are discovered, which were quite undiscoverable during life, because the blood did not enter the uriniferous tubules. If the amount of blood mingled with the urine be very small, the color of the latter is a peculiar dirty red when viewed by reflected light, while by transmitted light it is a pure red of greater or less depth. After standing for some time, a somewhat characteristic, slightly flocculent, brownish sediment is precipitated. When urine containing blood is exposed to the action of heat and nitric acid, the albumen of the serum of the blood coagulates. If the sediment be examined microscopically, blood-corpuscles are found, some of which are well preserved, while others are somewhat altered. There are also the casts above described, studded with blood-corpuscles, which are peculiarly characteristic of renal hæmorrhage. I strongly recommend *Heller's* test for blood as very simple and convenient, and by means of which the faintest trace of blood may be discovered. Heat the urine, then add caustic potash and heat anew. The phosphates are thus precipitated, taking with them the coloring matter of the blood, which imparts a dirty yellowish-red color to the sediment viewed by reflected light, and, when seen by transmitted light, gives a splendid blood-red color. Neither the coloring matter of the blood nor that of the bile is precipitated with the phosphates, so that coloration of urine which shows this reaction cannot be ascribed to the presence of the latter pigments. When the quantity of blood in the

urine is very large, it is of a dark or brownish red, and, after standing, forms a cake of blood at the bottom of the vessel. Not unfrequently, a partial coagulation of the blood takes place in the bladder, and the coagula thus formed can only be got rid of after great suffering on the part of the patient. Sometimes the blood coagulates while in the ureters. In such instances, the symptoms of renal colic, hereafter to be described, may arise, and long, worm-like coagula (casts of the ureters) may be voided.

The course of renal hæmorrhage necessarily depends upon the nature of the disease which causes the hæmorrhage. When induced by renal calculus, bleeding occurs regularly after every violent exertion. Hæmorrhage arising from the presence of a tumor, especially cancer, is usually very profuse and persistent. The bleeding which accompanies inflammation of the kidney and the infectious diseases, or which results from venous obstruction or scorbutus, is not often very severe. In the endemic hæmaturia of the tropics there are periodical profuse flows of blood from the kidneys.

In hæmorrhagic infarction, if there be hæmaturia at all, it is always slight. It is usually ushered in by a chill, and is attended by pain in the region of the kidney, and by violent sympathetic vomiting. The occurrence of such a train of symptoms—a chill, lumbar pain, vomiting, and hæmaturia—in a case of heart-disease, would warrant the diagnosis of a renal infarction, which otherwise generally escapes detection. Renal apoplexy is marked by similar symptoms, which, however, are of much greater intensity.

The results of renal hæmorrhage cannot be described in detail until after a more thorough discussion of the diseases whereon they depend. The hæmaturia arising from cancer, from renal calculus, and from the tropical disease, acts chiefly by producing exhaustion from loss of blood.

Treatment.—The main point in the management of a case of renal hæmorrhage is the treatment of any hyperæmia, inflammation, or other constitutional disease from which it proceeds. In most instances, when the main disease is susceptible of efficient treatment, the hæmorrhage does not require any especial attention. Sometimes, however, the danger of exhaustion arising from repeated attacks of profuse and persistent hæmorrhage, such as occur in cancer and renal calculus, may require relief. Cold, in the form of a bladder of ice laid over the region of the kidney, cold sitz-baths, or cold injections, should then be resorted to. Internal administration of styptics has been recommended, and the whole list has been given seriatim in many cases, each failing in its turn. Articles containing tannin, especially tannic acid itself, which is eliminated through the kidneys in the form of gallic acid, and thus is enabled to act directly upon the bleeding point, deserve the

greatest amount of confidence. Next to it, secale cornutum, or ergotine, may be given, but the doses must be large. The only effect to be expected from the preparations of iron is the beneficial action which they exert upon the deterioration of the blood.

CHAPTER III.

ACUTE BRIGHT'S DISEASE.

Etiology.—The name Bright's disease is generally applied to two forms of inflammation of the kidney. The first, which is the subject of the present chapter, is closely allied to croup of the larynx and air-vesicles, not only in its anatomical lesions—consisting of a coagulating exudation, containing epithelial cells, and often extravasated blood-cells, and which fills up and occludes the urinary tubules—but in its course, which is always acute, like that of the other croupous diseases above mentioned, and nearly always terminates either in recovery or death within a few days. It is rare for the disease to pass into the second form of Bright's disease, which we shall describe in the next chapter, under the name of "parenchymatous nephritis." This latter circumstance, indeed, seems to me to indicate that it is both right and practical to regard acute and chronic Bright's disease as independent and distinct affections. I attach little value to the term "croupous nephritis," applied to acute Bright's disease, in the previous editions of this work, as I must acknowledge that it is a matter of doubt whether the exudation which fills up and obstructs the uriniferous tubules consists of fibrin like the exudation of croupous laryngitis and croupous pneumonia, and as it cannot be denied that the epithelium of the uriniferous tubules takes a more active part in the nutritive disorders attending acute Bright's disease than is taken by the epithelial cells of the larynx and air-vesicles in croupous laryngitis and croupous pneumonia.

1. Acute Bright's disease is a frequent complication of scarlatina. There is a prevalent notion among the laity that a child who dies of dropsy, after scarlet fever, "has not been well taken care of;" and many an unhappy mother, who has lost her child from this cause, reproaches herself for years for having changed its linen too soon, or imprudently opened a door, and thus brought about her child's death. It is possible that chilling of the skin during scarlet fever may sometimes favor the occurrence of croupous inflammation, and may even actually produce it, but it is certainly not the case in the majority of instances. Besides the disturbance of the skin which it occasions, scarlatinous virus constantly induces disorders of the fauces and kidneys. In most

epidemics they are of a hyperæmic nature, and give rise to the well-known symptoms of catarrhal angina in the throat, and in the kidneys cause renal catarrh, as described in the foregoing chapter. There are malignant epidemics of scarlatina, however, which give rise to much graver disorder of these organs. Instead of simple catarrh, there is nearly always a diphtheritic inflammation of the fauces, and, instead of a simple renal hyperæmia, the urinary tubules are attacked by croupous inflammation. In such epidemics, many children die of dropsy who have received the best possible care, while many, who have been actually neglected, escape unscathed. The virus of measles, typhus fever, and the poison of malaria, may also induce croupous nephritis, but they are far less frequent causes of the disease than is scarlatina.

2. Acute Bright's disease arises during the typhoid stage of cholera, and by many is regarded as a constant complication, if not the actual cause, of this very common and obscure sequel of cholera. Although we cannot subscribe to this latter opinion, having witnessed the death of many patients from cholera typhoid, whose urine was abundant and free from albumen, yet the frequence of croupous nephritis, as a sequel to cholera, cannot be denied. It remains an open question whether the vascular engorgement and inspissation of the blood, which take place in the algid stages of cholera, induce obstruction of the renal capillaries from crowding together of the blood-corpuscles, and extravasation of plasma and blood into the tubules, or whether the inflammation of the kidneys, like the other inflammatory affections of the typhoid stage of cholera, be ascribable to infection of the blood. It, undoubtedly, is very rare for croupous nephritis, in healthy subjects, to proceed from contusions, the misuse of irritating diuretics, exposure to cold, or other unknown exciting causes.

Anatomical Appearances.—The anatomical alterations found *post mortem* after croupous nephritis are identical with those so admirably described by *Frerichs* as the first stage of Bright's disease, the "stage of hyperæmia and incipient exudation." The kidney is often enlarged to twice its proper size, and its surface is smooth. The tunica albuginea is opaque, injected, and is easily detached. The cortical substance, to whose swelling the increase in volume of the kidney is mainly due, is of a more or less dark-brown color, soft, and easily torn. When cut into, a bloody adhesive liquid bathes the face of the section. Both the superficial and deeper parts of the cortical substance are dotted with dark-red points. The pyramids also are hyperæmic and striped with red, and an opaque and often bloody liquid is usually found in the calices and pelvis of the kidney, which likewise are injected with blood. Microscopic examination does not

exhibit any important change in the texture of the kidney. The glomeruli, being overloaded with blood, are very distinct. Extravasations of blood are almost always found in the Malpighian capsules and tubules, which account for the blood-red points above alluded to. There are likewise extravasations between the albuginea and the tubules. The uriniferous tubules, especially those of the cortical substance, are filled with coagulated exudation. Upon microscopic examination of the liquid expressed from the cut surface of the diseased kidney, we find the cylindrical masses of exudation covered with epithelium and blood-corpuscles, forming casts of the tubules. The epithelium is not materially altered, only moderately swollen and clouded.

Symptoms and Course.—Sometimes croupous nephritis is ushered in by a rigor, followed by fever and a sharp pain in the region of the kidney. In addition to this, there is almost always more or less violent (sympathetic) vomiting; indeed, vomiting is a more constant token of incipient disease of the kidney than either fever or pain, and it is well to warn parents of children with scarlatina of the serious nature of this symptom, and to require them to seek medical aid should it arise. The patient feels a constant inclination to pass water, but is unable to expel more than a few drops with each effort. The suppression of urine may be so complete that the whole amount secreted in course of a day may not exceed an ounce or two. Its specific gravity is high. Sometimes, and for a short time, it may be of the color of pure blood; more usually it is opaque, and of a peculiar dirty reddish-brown hue, and looks as though it really contained dirt. Both urine and sediment have this dirty appearance, which alone, to the eye of an expert, is a tolerably sure sign of acute Bright's disease. There is a very large quantity of albumen in the urine, and, upon the application of heat and nitric acid, the half or even three-quarters of the liquid will coagulate. Upon microscopic examination of the sediment, we find large quantities of epithelium from the tubules and urinary passages, as well as many blood-corpuscles, and casts studded with blood-corpuscles. [Exact observation has taught us that the vascular walls, when inflamed, become permeable to substances which normally would not pass through them. Hence we may presume that it is not the effect of mere pressure upon the Malpighian tufts, but rather of some change in their filtering power which gives rise to the albuminuria in acute Bright's disease.] Dropsical symptoms set in, and in most cases the dropsy soon becomes very severe. The face, hands, legs, and scrotum swell up, and the skin is so tensely swollen that an impression made upon it by the finger is soon effaced. The dropsy of croupous nephritis, like that of parenchymatous nephritis, as we shall presently see, shows a great

tendency to shift its position, the swelling increasing in one part of the body, while it diminishes in another.

When the progress of the disease is favorable, the coagula which block up the uriniferous tubules are washed away, the urine becomes freer and more abundant, and the albumen diminishes. At the same time there is an abatement of the dropsy, which, in this disease, seems rather to depend upon suppression of the secretion of urine than upon that lack of the albumen in the blood which takes place in acute hydræmia. In the most fortunate cases, the disease may terminate in from eight to fourteen days, recovery being complete and without sequelæ. In very many instances the nephritis is accompanied by acute inflammation of the lung, pleura, pericardium, or peritonæum, and it is to one of these complications that the patient usually succumbs in fatal cases.

It happens much more rarely that croupous nephritis, instead of terminating in the above manner, gives rise to the so-called uræmic intoxication. It is easy to comprehend that, in consequence of the suppression of the urinary secretion, substances may accumulate in the blood which act perniciously upon the nutrition and functions of the various organs. It used formerly to be supposed that the urea, which is the most abundant of the solid constituents of the urine, and which is the best known of all its organic ingredients, by accumulating in the blood, induced convulsions, coma, and ultimate palsy of the entire nervous system; and when such symptoms accompanied suppression of urine, they were called uræmic symptoms, or uræmic poisoning. *Frerichs* afterward supposed that this toxic effect was due to the presence of carbonate of ammonia, resulting from decomposition of the urea, rather than to the urea itself. This theory, however, cannot by any means be regarded as proved, and we must acknowledge that we are unacquainted with the excrementitious material retained in the blood, which exerts so pernicious an influence upon the organism in cases of suppression of urine. However, notwithstanding the rarity of uræmic poisoning in croupous nephritis, yet it is of great importance, as regards both the prognosis and treatment of the disease, not rashly to ascribe any convulsions or stupor which may arise to inflammation and exudation in the brain. Cases occur in which the convulsions and coma subside as the free secretion of urine is reëstablished, and the attack terminates favorably. (For further details of the so-called uræmia, and of the frequent dependence of cerebral symptoms upon œdema of the brain, see Chapter IV.) I should finally state that cases of croupous nephritis occur in which the disease improves somewhat, but does not subside completely; the albuminuria continues, and the

character of the acute croupous nephritis (acute Bright's disease) runs into that of chronic diffuse nephritis (chronic Bright's disease). I have never seen such a case, and they must be very rare.

Treatment.—In recent cases, and in tolerably robust subjects, it is advisable to resort to local depletion over the region of the kidneys, by means of leeches or cups. The effect of the first application will decide as to the propriety of its repetition in case of recurrence of the symptoms. General blood-letting should be rejected, as liable to aggravate the already existing tendency to deterioration of the blood. The use of calomel and other so-called antiphlogistics is equally improper. Warm baths, followed by envelopment of the body in woollen blankets, are much preferable to the internal exhibition of diaphoretics. In treating of chronic Bright's disease, we shall go more into detail upon this subject, and upon the brilliant results which are sometimes obtained from diaphoresis. If the bowels be confined, a few powerful doses of drastic medicine should be given—jalap, senna, or even colocynth. The profuse serous transudation into the intestine caused by these medicines may have a beneficial effect upon the dropsy ; but it sometimes happens that the patient has a violent diarrhœa, and yet the dropsy continues to increase rapidly. Mineral waters containing carbonic acid are the most suitable beverage. We should not be too sparing in their administration, as the increase of pressure in the glomeruli, and the augmented transudation, may assist in washing away the obstructing coagula. On the other hand, drastic diuretics are contraindicated, owing to the inflamed condition of the kidney. During convalescence, and in tedious cases even prior to convalescence, the tendency to deterioration of the blood must be combated by the administration of quinine and iron, and a plentiful supply of albuminous food.

CHAPTER IV.

CHRONIC DIFFUSE NEPHRITIS—CHRONIC BRIGHT'S DISEASE.

[Pathology and Etiology.—The following synopsis may serve to give the reader an insight into the present state of knowledge regarding Bright's disease. We have long made use of *Frerichs's* classification of the anatomical stages of the disease : 1st, the stage of hyperæmia ; 2d, that of exudation and of commencing conversion of the exudation , 3d, the stage of retrogression and atrophy. But it has been found that the first of these conditions properly belongs only to acute diffuse nephritis ; so that for chronic nephritis there

remain only the stage of inflammatory swelling and that of atrophy and shrinkage. But even the connection between these two stages has been denied by certain authors—*Johnson* and other English writers—who maintain that these conditions, instead of being phases of one and the same disease, are the products of totally different pathological processes.

The very great diversity of opinion regarding the histology of Bright's disease may be summed up as follows: For some authors the essential primary lesions in all forms of the malady originate in the renal epithelium (*Johnson*); for others, the disease begins only in the interstitial connective tissue (*Traube*). Others again believe that it may commence in both or either of these tissues. Hence they recognize an *intertubular* or *interstitial*, and an *intratubular* or *parenchymatous* form of the malady; but they admit the coexistence of the two forms. *Johnson* first pointed out a hypertrophy of the muscular coat of the small arteries, not only of the kidney, but of other organs, and showed that under such circumstances the epithelium cannot remain intact; and he traces back the hypertrophy of the left ventricle of the heart, so often met with in Bright's disease, to the greater resistance opposed in that affection by the hypertrophied vessels. Latterly there has been great controversy upon the views of *Gull* and *Sutton*, according to whom, in the contracting form of the disease, the primary lesion is not in the kidney at all, but the atrophied kidney is merely one of the lesions which accompany a general disorder of the minute arteries and capillaries, and which consists in a deposit of hyaline lymph in the adventitia of the arteries (seldom in the intima), and in the outer coats of the capillary walls. It must be admitted that shrinking of the kidney and degeneration of the arterial walls often coexist; but further research has shown that such coexistence of the two lesions is by no means constant.]

In Bright's disease the epithelium of the uriniferous tubules exhibits the alterations which we have repeatedly described as characteristic of all parenchymatous inflammations. Its cells first increase considerably in bulk, through imbibition of an albuminous liquid; their contents then undergo fatty metamorphosis, by which the epithelial cells gradually become converted into cells of fatty granules. Finally, the cell-membrane perishes, and the fat-globules emerge free into the urinary tubules.

While these are the essential changes which the kidney undergoes, in the majority of cases coagulating exudations are also formed in the tubules, and in many instances proliferation of the interstitial connective tissue occurs. Atrophy of the kidney, which sets in

afterward, is the natural and necessary consequence of the inflammatory process above described.

Bright's disease is a very common affection. Predisposition to it is far less in childhood than in more advanced life. Men are attacked by it somewhat more frequently than women; persons of debilitated and depressed constitution more readily than those who are robust. Hence the poorer part of the community are more afflicted by the disease than the well-to-do class, being more exposed to the evils which produce it.

1. Chief among the predisposing causes of Bright's disease is the temporary, and in a still greater degree the continual exposure of the skin to the effect of cold and moisture. This accounts for the great frequence of the disease in England, Holland, Sweden, and on the German coasts; and not only upon that of the North Sea, as *Frerichs* has assumed, but also upon the eastern shore. In the very moderate number of beds in the Greifswald clinic, there used always to be many cases of Bright's disease.

2. It would seem that the misuse of irritating diuretics and the incautious exhibition of cubebs and copaiba may sometimes lead to Bright's disease, although, perhaps, this does not occur very frequently.

3. On the other hand, the abuse of ardent spirits unmistakably plays a most important rôle in the etiology of the disorder, so that Bright's disease appears almost as frequently among hard drinkers as does cirrhosis of the liver. We have no physiological explanation of this circumstance; but as recent researches have proved that alcohol taken into the system is not all consumed in the blood, as used formerly to be supposed, but that at least a portion of it, passing through the urinary organs, is eliminated from the system unaltered, it is conceivable that the alcohol may act locally upon the kidney, just as we have shown it to act in cirrhosis of the liver.

4. Bright's disease very frequently is associated with tedious suppuration, with caries and necrosis of the bones, the surgical wards of the hospitals always furnishing a rich contingent of this malady; although the latter causative agents lead to amyloid degeneration of the kidney with equal if not greater frequence. The connection between these exhausting affections and Bright's disease is obscure. However, just as in other cachectic conditions, so the appearance of inflammation in the most diverse organs is so common an occurrence in these depressing maladies, that it becomes a question whether nephritis holds a closer relationship to such conditions than is held by pleuritis, pericarditis, peritonitis, and the like.

5. Finally, Bright's disease often accompanies conditions of dyscrasia, resulting from gout, rachitis, syphilis, scrofula, and malarial cachexia, in which, however, besides the inflammatory degeneration, the lardaceous metamorphosis hereafter to be described is often observable.

I agree with *Traube*, that it is improbable that simple obstructive hyperæmia from disease of the heart and the like gives rise to inflammation of the kidney; and I believe that, hitherto, there has been a great deal of confusion of this malady with that described in the preceding chapter. Nor can I regard pregnancy as one of the remote causes of Bright's disease. The albuminuria so common among pregnant women is not generally a result of inflammation, but is rather due to a parenchymatous degeneration of the kidney, to be described in Chapter VIII.

[Malarious poison predisposes to Bright's disease, and, according to *Bartels*, is one of the most prominent of its causes. But an albuminuria arising during an acute febrile disorder has never been observed to terminate in chronic renal disease. On the other hand, chronic suppuration with continued fever may be followed not only by amyloid degeneration, but also by chronic inflammatory swelling of the kidney.

Those observers who regard parenchymatous and interstitial nephritis as distinct diseases naturally ascribe each of them to different causes. The causes of that form which develops so insidiously and so very gradually in the interstitial substance are extremely obscure; and *Bartels* remarks regarding these so-called "*contracted kidneys*," which he looks upon as a separate disease, that in scarcely one of his cases could his patients even guess at any cause for their malady over and above the influences which always more or less beset them. English writers ascribe shrunken kidney largely to gout and to lead-poisoning. *Dickinson* states that twenty-six out of forty-two persons with lead-poison had died of shrunken kidney. While this form of renal disease is more common in advanced age, the other occurs more often between the ages of twenty and forty. In childhood it is rare, if we except cases of acute diffuse nephritis following scarlatina.

Pathological Anatomy.—According to the present views of many, the term chronic Bright's disease must be regarded as embracing two distinct forms of nephritis, the *interstitial* and the *parenchymatous*. The former, according to *Klebs*, begins with intense hyperæmia, with exudation of a lymph-like liquid throughout the whole cortical substance of both kidneys, which dilates the lymph-spaces of the interstitial tissue, and is accompanied by a constantly

increasing emigration of the white blood-corpuscles. Under favorable conditions these cells may undergo fatty degeneration, and a complete resolution of the process may ensue; otherwise they may convert themselves into fibrous tissue, causing permanent thickening with or without shrinkage of the intertubular substance.

In such a case as this, the process drags on for years; and we find after death both kidneys shrunken, sometimes even to a third of their normal size, and the capsule thickened and opaque, and firmly adherent to the parenchyma. The surface of the organ is of a dull brownish-red, here and there of a dirty-yellow hue. The atrophy being complete in some parts of the kidney and less advanced in others closely adjacent to the first, the surface becomes studded with small nodules, some of the size of a pin's head, and some as large as a pea. Hence the term *granular kidney.* Such a kidney is very hard and tough. Upon section, the cortical substance is often found to be so much wasted that the secreting parenchyma only forms a narrow rim around the pyramids, while the latter are often but little reduced in size, being but sligthly affected by the disease. Certain whitish stripes sometimes observed correspond to the straight tubules which have become obliterated for want of supply of secretion. The general diminution of the kidney, with atrophy of many of its glomeruli and tubules, is always in the main ascribable to the compression exerted by the contracting newly-formed connective tissue; but other factors also contribute to this, such as wasting of the epithelium and urinary tubules. It is sometimes found that the process of thickening of the tissues is much more pronounced in the Malpighian capsules and their vicinity; or, on the other hand, the greatest change may be in the tunica propria of the tubules and in the connective tissue between them. Accordingly, *Traube* distinguishes two forms of interstitial nephritis, the circumcapsular and the intratubular, each of which presents certain peculiarities from a clinical point of view. Not unfrequently, in the cortical substance we find numerous small cysts having clear contents, and varying in size from that of a millet-seed to that of a pea. These probably are the product of constriction and dilatation of parts of a uriniferous tubule, particularly of one of the convoluted tubes.

The chronic parenchymatous form is regarded by some observers as an inflammation which occurs exclusively and entirely in the secretory cells of the tubules. We do not hold it proved that there is such a form, and think that there may be confusion with the so-called "opaque swelling" of the kidney epithelium (Chapter VIII.). But we find that in some cases the elements of the tubules take

little part in the inflammatory process which is going on in the intervening tissues, while in others it is the prevailing seat of the disorder. We believe that it depends upon the degree of the inflammation whether the secretory or merely the interstitial part of the organ shall be invaded. We shall see by and by that chemical observation also teaches that cases of a very tedious insidious course betoken an interstitial disease, while, on the other hand, chronic cases of a more active and less protracted form indicate the existence of the parenchymatous disease. In the acute diffuse nephritis above described, the implication of the renal parenchyma proper is most apparent both from a clinical and anatomical point of view. If we trace back the difference between the parenchymatous and interstitial forms to a gradual variation in grade of the inflammation, the existence of many intervening forms will be easy of comprehension.

In the chronic parenchymatous form, the kidney, in consequence of swelling of the epithelium and of deposits of exudation in the tubules, becomes very soft and swollen, sometimes to double its normal size. The vessels of the cortical substance are bloodless (in the cadaver at least), the capsule is opaque and easily detachable, and beneath it the renal surface looks pale or yellowish, except where one or two venous trunks remain filled with blood. Upon section it is seen that the enlargement is all due to swelling of the cortical substance, which is sometimes an inch thick ; and that the pyramids take no part in the pallid coloring of the cortical substance, but often form a marked contrast with it by their deep-red hue. Here and there a Malpighian body still remains like a red point on the pale field. The chief seat of the morbid process is in the convoluted tubules of the cortical substance, while the straight tubules of the pyramid rarely, and in but small degree, exhibit the exudation which causes the swelling and softening of the cortical substance. The cylindriform deposits, which occlude and render varicose many tubules, consist partly of swollen fatty epithelium, partly of lymphatic elements (pus) and blood-corpuscles, but especially of hyaline or granular cylindrical masses.

Many observers assume that, after a certain duration, the enlarged kidney gradually shrinks and atrophies, and suppose the atrophy to arise from the perishing of the glomeruli, by pressure of the intracapsular exudation, and the collapse of their tubules which have lost their epithelial lining. Even admitting the occurrence of such obliteration of some of the tubules, it may be questioned whether atrophy of the kidney can really be brought about through such a process alone, and without the concomitant thickening of

the intertubular tissue by interstitial nephritis. In fact, even the eminent authorities who believe in the distinct nature of the two forms of nephritis, nevertheless admit the possibility of the existence of many intermediate or combination forms between the two. (*Bartels.*)

To conclude our description of the pathological anatomy of this affection, it may be well to remark that in many instances the morbid changes which take place in the kidney are by no means easy of recognition by the naked eye. When the inflammation is of moderate extent, we often find merely a few pale-gray streaks lying in the renal tissue, which in all other respects looks perfectly healthy. Sometimes, when the disease is diffuse and scattered, it would be impossible without the aid of the microscope to determine with certainty its presence in the tubules and interstitial tissue. The numerous consecutive changes which chronic diffuse nephritis is apt to bring about in other organs and tissues will be duly mentioned when we come to discuss the symptoms of Bright's disease.]

Symptoms and Course.—Pain in the region of the kidney, which has been reckoned by most authors as among the most constant symptoms of morbus Brightii, according to my observation, is wanting in the majority of cases throughout the entire course of the disease. It is true, that if we press with great force upon the kidney, the patients complain that the procedure is uncomfortable and distressing, but we shall hear a like complaint from well folk, whom we may subject to similar infliction. It is as unusual for the attention of the patient to be called to the grave nature of his disease by any marked diminution in the amount of urine which he passes, as it is for him to suffer pain in the renal region. After the dropsy and the albuminuria have placed the diagnosis beyond a doubt, most patients, if asked whether they have passed too little urine in the course of their disease, will not only deny it, but will even declare that throughout the entire duration of their dropsy they have made a great deal of water. Such an account as this, from a patient with chronic dropsy, is in itself suggestive of the probable dependence of the dropsy upon chronic renal disease. On the other hand, if a patient assert that his dropsy has developed gradually, and that since its commencement he has always passed remarkably little urine, there is a certain amount of presumption that the dropsy is of cardiac or pulmonary origin, and that it does not proceed from disease of the kidney. However, we must not ignore the fact that such statements from patients, as to their passing an unusual quantity of water, are often the result of a delusion upon their part. An inclination to pass water frequently, a symptom due to sympathy of

the urinary bladder, and common both to chronic and to acute Bright's disease, but which is never very severe, impresses the patient with the idea that, in his frequent acts of micturition, he has discharged a large quantity of water. Accurate measurement of the amount passed in twenty-four hours shows that, in many cases, it does not quite reach the normal flow. In others it is normal; while in others, again, the proper quantum is really exceeded. A considerable diminution of the normal secretion or actual suppression of urine seldom occurs, and then merely forms, as it were, a short episode in the disease. This peculiar behavior of the urinary secretion, in chronic Bright's disease, is altogether enigmatical. The slighter degree of suppression, which is the most frequently observed, admits of the easiest explanation; as the obstruction of numerous tubules by swollen and degenerated epithelium would obviously impede the outflow of the urine, and the compression of many of the glomeruli must effect a diminution in the amount of urine secreted. But how shall we explain the fact that, in many instances, in spite of this hinderance to the discharge, and in spite of the limitation of the secreting surface, the flow of urine still remains normal, or even is abnormally profuse? How account for the fact that the increase in this secretion is peculiarly common in the third stage of the disease, at a period when the kidney is atrophied, and when many of its tubules and Malpighian capsules have collapsed and wasted away? We admit that the hypertrophy of the left ventricle of the heart may perhaps assist in augmenting the secretion of urine by increasing the pressure within the glomeruli, which still remain intact, thus hastening the filtration of liquid through them; but its influence is by no means to be so highly rated as to suppose that the absence of many defunct glomeruli can be more than compensated for by increase of internal pressure upon those which remain. Nor does the collateral fluxion to the remaining glomeruli, induced by obliteration of the blood-vessels in the affected portion of the kidney, do more than to explain why there is not a material decrease in the secretion of urine, and by no means accounts for its augmentation. There is some probability that privation of the blood of its albumen may have some effect in increasing the secretion from the kidney. As is well known, a liquid will pass more freely through an animal membrane from a dilute solution of albumen, than from a solution which is more concentrated, the pressure being the same. But even this does not seem to account for the increased production of urine which we so often observe in the third stage of Bright's disease.

Although, as before said, neither pain in the region of the kidney, nor any unusual flow of urine, calls attention to the grave disease which is going on, yet its recognition is no longer difficult, since it has

become the practice of the better class of physicians to make careful examination of the urine. Hospital patients do not generally apply for aid until the dropsy has appeared. Exclusion of other causes of dropsy will establish a strong presumption that we have a case of Bright's disease to deal with. Examination of the urine places the diagnosis beyond a doubt. In private practice, the observant and experienced practitioner will have recognized the disease before the dropsy sets in. The history of the case is almost invariably as follows: The patients have long remarked a failure of their strength, and a pallor and anæmic aspect of their skin, and visible mucous membranes. As all their functions are apparently normal, they are unable to account for this paleness and debility. The physician, after careful examination of all other organs, can find no appearance of disease to which the loss of strength and impoverishment of the blood can be ascribed. He examines the urine, and finds it to be loaded with albumen, and the symptoms are accounted for. No elaborate demonstration is required to show that, in addition to the other expenditures of the blood, a daily loss from the blood of large quantities of albumen, which may amount to from twelve to twenty grammes in the twenty-four hours, cannot be made good by the daily supply of nourishment; or, in other words, a person subject to a daily drain from his blood of from twelve to twenty grammes of albumen necessarily becomes pale, bloodless, and enfeebled. As an examination of the urine reveals the existence of the disease before the occurrence of the dropsy as well as after it, it will be well to give a more detailed account of the characteristics of the urine secreted in parenchymatous nephritis. It is generally of a pale-yellow color, and often exhibits a somewhat opalescent reflection. As it is more viscid than common urine, on account of the albumen which it contains, it is more easily made frothy than urine free from albumen, and the froth lasts longer. When there is no intercurrent febrile disease, its specific gravity is remarkably low, and may sink to 1005. This is principally on account of a decrease in its urea, and in a lesser degree owing to a diminution of the salts, especially the alkaline chlorides. The reduction in the amount of urea cannot at first be ascribed to its retention in the blood. It would seem rather that, just as in other hydræmic conditions in which the urine is abnormally light, the metamorphosis of material in the body is going on more slowly than is natural, thus retarding the production of urea. The explanation of *Schmidt* as to the decrease in the saline constituents of the urine, especially its alkaline chlorides, is less satisfactory, namely, that the saline ingredients of the blood augment as its albumen diminishes, and *vice versa*. When the patients are already dropsical, the transfer of the chlorides into their dropsical effusion is a

phenomenon of far greater importance. The significance of this circumstance consists in the fact that, as long as the dropsy keeps increasing, the saline contents of the urine are very small, but whenever any rapid diminution of the effusion takes place, the salts are eliminated into the urine more freely, much more freely, indeed, than under normal conditions.

If a portion of the urine be heated, after addition of a few drops of acetic acid, in case its reaction should be alkaline, or if nitric acid be added, the albumen coagulates. According to *Frerichs*, its quantity ranges from about 2.5 to 15.0 p. M. This presence of albuminuria, which usually persists throughout the whole course of the disease, and only disappears now and then, for short periods, unfortunately cannot be satisfactorily accounted for. One might be led into mistaking the albumen and the exudation cylinders for the products of inflammation, excreted from the free surface of the tubules, were it not that, in other and non-inflammatory diseases of the kidney, the urine contains both tube-casts and large amounts of albumen. I believe the presence of albumen in the urine to depend upon the destruction or degeneration of the epithelium. That normal urine should not contain albumen is confessedly extremely perplexing to the physiologists. They are almost forced to suppose that the albumen does transude into the kidney, together with the water and salts; and they are reduced to the hypothesis that its absence from normal urine is in some way connected with the epithelial lining of the uriniferous tubules, the transuded albumen either becoming assimilated for the nutrition of the epithelium, or else its diffusion into the tubules, receiving some other modification, as yet unknown to us, from the epithelium. The observation that albuminuria exists in all diseases of the kidney, in which its epithelium is either degenerated or destroyed, fully confirms this physiological hypothesis.

After the urine has been allowed to stand for a while, a light, whitish, flocculent precipitate falls to the bottom of the vessel. If this sediment be placed under the microscope (for this purpose it is best to let it deposit in the bottom of a pointed champagne-glass), the well-known casts are found. At the commencement of the disease they are covered by epithelium, in a state of fatty metamorphosis; at a later period, they seem quite bare, or are merely covered with granules and globules of fat. Besides this, the sediment contains common epithelial cells from the urinary passages, and smaller round, slightly-granular cells.

[By many the so-called urinary cylinders are supposed to be a product of the blood; others ascribe their origin to alteration of the epithelium. They probably originate in various ways. The

cylinders should be distinguished from those so-called epithelial tubes formed by separation of long pieces of the lining of the straight tubes, and recognizable under the microscope as oblong rolls of epithelial cells. Such casts appear in the less serious diseases of the kidney; hence their temporary presence warrants a favorable prognosis unless gainsaid by other signs. The cylinders proper belonging to the graver malady are solid plugs of exudation which have filled portions of tubes. There are two typical forms of them, with intermediate varieties: 1. Hyaline casts, which, though well defined, are transparent as glass, and hence are apt to be overlooked unless foreign matter adheres to them; 2. Granular casts, more or less closely studded by minute fat-globules. Hyaline casts are probably formed by the exudation of a fibrogenous substance, which becomes fibrin within the tubules. Some ascribe their origin to colloid metamorphosis of the epithelium. Another special modification of the hyaline cast is the waxy cast, which proceeds from the former, and is to be distinguished from it by its greater consistence, its yellowish color, and its more distinct outline. By many, granular casts are supposed to be the product of fatty metamorphosis of hyaline cylinders; others regard them as the result of croupous degeneration of the tubular epithelium. Other objects are often found adhering to the casts, such as pus, white blood-corpuscles, blood-disks, degenerated epithelium, and clusters of fat-globules, vestiges of broken-down cells; likewise the various crystals which are found in the urine. All the above-named objects are also found free in the urine of Bright's disease. During an inflammatory relapse an exceptionally large number of casts will appear in the urine; otherwise they are not numerous, and sometimes can only be found after filtering the urine and examining the dregs. Urinary casts are not a pathognomic sign of Bright's disease, for they are found, as we shall see, in other renal affections; nor can we positively determine the form or stage of the disorder from the character of the casts. Nevertheless, the discovery of many casts, both hyaline and granular, with here and there an adhering lymph-cell or granular epithelial cell, favors a diagnosis of an active progressive inflammation (a parenchymatous nephritis); while, if the casts are few, small, and hyaline, or if they are absent altogether, the urine being profuse, clear, and scant of sediment, we may suppose the disease far advanced, and that the kidney is shrunken (interstitial nephritis).]

Dropsy is one of the most characteristic signs of Bright's disease, excepting in a few rare instances, in which it has been absent throughout the entire course of the malady. It generally begins as anasarca.

At first the face and feet swell, the upper extremities, abdomen, and scrotum not becoming affected until a later period. The œdema often shifts its position in a peculiar manner, so that at one time the face or upper extremities may be the more swollen, at another the feet, abdominal walls, or scrotum, while the tumefaction subsides in the region at first affected. Moreover, if the patient be out of bed, the feet generally show the greatest amount of swelling in the evening, while in the morning the feet are smaller again, and the swelling involves the hips, back, and hands. The more slowly the anasarca develops, so much the more does the skin lose its elasticity, and so much more slowly is the imprint of the finger effaced from the œdematous surface. The addition of ascites and hydrothorax to the anasarca does not take place until a more advanced period. In one exceptional instance, I have witnessed the appearance of hydrothorax and œdema of the lungs early in the disease, where previously there had only been a slight œdema of the integument, a condition of apparent sècurity thus suddenly becoming one of great danger. In cases which advance rapidly, the dropsy may attain great magnitude in a few weeks. I have seen a patient who weighed a hundred kilogrammes (i. e., two hundred and twenty pounds), who affirmed that, eight weeks before, he did not weigh fifty. Such extreme dropsical swelling, from the strain which it exerts, may be the cause of inflammation and gangrene of the skin, especially of the scrotum and labia majora. In the worst cases, the skin often bursts at several points, and the liquid trickles copiously from the rents.

The pathogeny of the dropsy of parenchymatous nephritis is exceedingly difficult of explanation. As is already stated, the dropsy often develops in the midst of a copious excretion of liquid from the kidneys, and hence cannot be ascribed to any increase of pressure upon the veins of this organ, although this must be regarded as the main cause of the dropsy of acute croupous nephritis, in which disease there is always a suppression of urine. It is true that, during any check to the secretion of urine which may occur in this disease, the dropsy makes rapid headway; and when the urine is scanty throughout the entire course of the malady, the dropsy soon becomes excessive, and the malady runs a subacute course. There is no doubt but that a "hydræmic crasis," a lack of albumen in the blood, favors the occurrence of dropsy. Owing to the continual loss of albumen, which the blood is suffering, in Bright's disease, a liquid which contains but little albumen flows through the capillaries of the system. Hence, an abnormal transudation takes place from the capillaries into the interstices of the tissues, and hence, too, the amount of serum returned to the veins is abnormally small. It is not to be doubted that the ab-

sorption of liquid from the interstices of the tissues into the vessels is more active, in proportion as the difference in concentration in the liquid contained in the vessels and that without them is greater. Now, as, in parenchymatous nephritis, this difference is abnormally small, it follows, of course, that not only more liquid should leave the vessels, but that less should return to them. I have observed cases at my clinic which confirmed the correctness of this in the most striking manner. A girl, who had been suffering for a year from parenchymatous nephritis, stated that, some time before the appearance of the œdema, she had felt exceedingly dull and miserable. Being considered plethoric, she was advised to get bled. A week after the blood-letting, the first symptoms of anasarca showed themselves, and since then have never entirely disappeared. It may be inferred in this case that the hydræmia, which, though extant in moderation, still had not as yet produced dropsy, was so much aggravated by the blood-letting as to cause the dropsical symptoms to appear. In another case, the patient became anasarcous after a hæmorrhage, but the anasarca afterward disappeared for a while, and set in anew after the establishment of profuse suppuration. Nevertheless, hydræmia is not the sole cause of Bright's disease, and probably not even its chief cause. Dropsy, as severe as that seen in Bright's disease, is hardly ever observed in any other form of hydræmia. It often occurs early, but does not keep pace with the privation of the blood of its albumen. The peculiar manner in which the œdema shifts from one region to another is another argument against its dependence upon simple hydræmia. If we abstract blood from an animal, and inject a corresponding amount of water into its veins in its stead, the animal does not become dropsical. Finally, the dropsy is so very often accompanied by attacks of inflammation as to indicate that, besides the thinning of the blood, there is another source both of the transudation and exudation, consisting in some disorder of the tissues as yet unknown to us.

It sometimes happens that the continual aggravation of the symptoms above described, and the excessive dropsy, which finally may involve the serous sacs and alveoli of the lungs, cause death without further complication. In most cases, however, remissions occur, in which the condition of the patient improves, the albumen in the urine diminishes, and the dropsy subsides. After a while he grows worse again, perhaps once more to improve at a later period, and thus the disease will fluctuate. In such protracted cases the above symptoms are seldom the only ones, but are accompanied by others which arise in part as complications of the main disease, and in part are immediate consequences of it.

Of these, inflammation of the lungs, of the pleura, pericardium, peritonæum, and meninges, deserve the first mention, as they are extremely frequent complications of Bright's disease, and because it is of these intercurrent affections that the patient most frequently dies, far more frequently, indeed, than of the so-called uremic symptoms—to be described presently—or even of excessive dropsy. Such inflammations of the lungs, pleura, etc., do not differ from similar inflammations in other conditions of anæmia. The patient seldom succumbs at once to the first attack; and we not uncommonly find vestiges of previous inflammation, such as adhesions of the pleura, pericardium, and peritonæum, besides the marks of the final seizure. Parenchymatous nephritis is often complicated by catarrh, particularly by catarrh of the bronchi and intestine. There is nothing peculiar about the former, although in some cases the secretion is tolerably copious. The latter, however, is almost always characterized by a very abundant serous transudation, and by its extreme obstinacy. It would seem that the same cause which induces the escape of such large quantities of liquid into the subcutaneous connective tissue also gives rise to this immense transudation upon the free surface of the bronchial and intestinal mucous membrane. Nevertheless, since the dropsy is not invariably accompanied by catarrh, it must be admitted that its origin is somewhat obscure.

According to my experience, chronic œdema of the lung is of very common occurrence in Bright's disease. It gives rise to great dyspnœa, to a tormenting cough, and, at the climax of the coughing-fit, not unfrequently induces vomiting. I have had repeated opportunity of observing, whenever the vomiting caused the patient to eject much secretion, that his breath became freer for a while, his cough ceased, and that the fine subcrepitant râles subsided. Paroxysms of so-called urinous asthma, which are said to occur in Bright's disease, are probably dependent in a great degree upon œdema of the lung.

Many patients with parenchymatous nephritis suffer from disease of the heart. Besides the adhesions of the heart and pericardium from former pericarditis, and the valvular derangement resulting from endocarditis, none of which are uncommon in Bright's disease, we very often find a hypertrophy of the heart, and more especially a hypertrophy of its left ventricle. *Traube* has advanced the theory that this hypertrophy is a result of derangement of the circulation of the kidney, which he claims should augment the labor of the heart. This theory is disputed by *Bamberger* and others, who reply that the hypertrophy develops in a stage of the disease when no obstruction of any importance to the circulation of the kidney exists. A more extensive collation of facts will be necessary to decide

this disputed point; but, at all events, enormous hypertrophy of the heart sometimes occurs even in the second stage of Bright's disease, and assuredly the circulatory disturbance of the kidney is not the sole cause of it. As is well known, the signs of the enlargement are not very striking, but, by paying attention, we can often detect an augmented heart-shock, or, in its absence, hear remarkably loud cardiac sounds.

In a great number of cases, the symptoms of the so-called uræmic poisoning do not appear at all throughout the entire course of the complaint. Sometimes they develop slowly and gradually; sometimes they come on very suddenly. At times (but not always), the attack is preceded by a decrease in the secretion of urine, and in rare instances it has happened that, during or immediately prior to the appearance of the uræmic phenomena, the normal flow of urine has been largely exceeded (*Liebermeister*). It is a suspicious sign when patients complain of severe headache, and become languid and apathetic, and still more so, if these symptoms be accompanied by vomiting, which occasionally is so very obstinate as to awaken apprehension of serious disorder of the gastric mucous membrane. All these symptoms may subside again, without evil consequence; in other cases, however, the drowsiness increases to a deep stupor, or convulsions of an epileptic or more rarely of a tetanic character may arise. Even when the convulsions have not been preceded by drowsiness, they are usually followed by a condition of deep coma with stertorous breathing. The fits recur at longer or shorter intervals, the stupor meanwhile continuing to grow deeper; and the patient may finally succumb to general paralysis. It is not at all rare, however, for the fits gradually to become less frequent, the intervening stupor less profound, and for the signs of "uræmia" to disappear, perhaps not to recur for weeks or months. In a previous section, we have already acknowledged that we are unacquainted with the nature of the poison causing uræmic intoxication. There is a second difficulty in explaining the uræmia of parenchymatous nephritis, since certain well-attested cases of uræmic poisoning have been observed, in which there was no suppression of urine. If the urea and other material to be eliminated from the blood pass into the tubules by a process of pure endosmosis, it remains inexplicable how these materials can accumulate in the blood when the urine is secreted freely; hence we must assume that the epithelium of the urinary tubules has some important influence over the secretion of urine, and therefore that its disease or death may occasion an abnormal state of the blood, even though the kidneys continue to discharge a sufficient quantity of liquid. Moreover, I think that it would be going too far to attribute all the grave nervous symptoms which

arise in parenchymatous nephritis—the headache, the convulsions, the coma, etc.—simply to a poisoning of the blood; and for many cases at least I agree to the equally one-sided views of *Traube*, according to whom the so-called uræmic symptoms depend upon œdema of the brain and cerebral anæmia. Since the first appearance of my text-book, I have been much gratified at the steady advance made by the doctrine which I propounded long ago, that the symptoms of the so-called cerebral pressure, due to encroachment upon the cavity of the cranium, whether by a depression of the skull, hæmorrhage, tumor, abscess, inflammatory exudation, or serous transudation, all depend upon an arrest or obstruction to the flow of blood to the ganglion-cells and nerve-fibres of the brain. But, in spite of the experiments of *Munk*, I hold it to be unproved, and even improbable, that the acute œdema of the brain in Bright's disease should have an origin different from that of œdema of other regions, or that it should be ascribable to an increase of pressure within the cerebral arteries. Moreover, it seems to me to be extravagant to endeavor to ascribe all cases of so-called uræmia to compression of the cerebral capillaries, and to anæmia of the brain. My position in this question is as follows: In chronic parenchymatous nephritis, various organs are subject to œdema, the precise cause of which is unknown. It is characteristic of this œdema, that it shifts its position. It may attack the lungs at any period, either early or late in the disease, sometimes causing death, and sometimes subsiding again after a short duration. In a manner precisely similar, and for the same unknown reason, the brain may become the seat of an acute or subacute œdema, to which many succumb, while in others the œdema changes its position, and the patients are restored to a state of tolerable comfort for a period of variable duration. Many cases of so-called uræmic intoxication, but by no means all, are the result of œdema of the brain, and consequent anæmia of the cerebral capillaries. We may infer that an attack of this kind depends upon such an œdema, and not upon blood-poisoning: 1. When the seizure takes the form of deep coma, with intercurrent eclamptic spasms. 2. When, at the time of its occurrence, the secretion of urine is normal or increased. 3. When the attack is accompanied by marked œdema of the face. 4. When the carotids pulsate strongly during the attack. As we shall see directly, this is a valuable but often ill-appreciated sign of repletion of the cranial space with blood, and of impediment to the exit of the blood from the same.

[The temperature in uræmia may be either increased or diminished. According to *Bourneville*, the average temperature is reduced, but rises during the fits, and remains high during the intervals. In fatal cases it is very high, but it sinks gradually to the

normal when the outlook is favorable. According to prevalent modern views, the cause of the uræmic symptoms is due to insufficient elimination by the kidneys of certain products of the body which, when sufficiently concentrated in the tissues, have a poisonous action upon them, especially upon certain parts of the brain. This poisonous effect is exercised chiefly by the urea and its product of decomposition, carbonate of ammonia.

According to *Rosenstein*, however, poisoning by carbonate of ammonia alone induces but one of the trains of symptoms, that of epilepsy, while the agent which produces the uræmia occasions not only epilepsy, but coma, convulsions, and delirium. Moreover, we must remember that in cases of uræmia, even when the presence of carbonate of ammonia in the blood has been demonstrated, the dependence of the uræmic symptoms upon its presence has not been proved. The quantity of urea excreted in the course of a day is generally diminished in Bright's disease. This diminution of the daily quantum of excretion is often considerable at the very time when the secretion of urine is also reduced ; while in patients with extreme polyuria, whose health is as yet unshaken, the quantity of urea excreted, in spite of the small percentage contained in the urine, may even exceed the normal. If the daily excretion of urea is very small, we are justified in fearing that the disproportion between production and excretion may lead to uræmia.]

In many cases of Bright's disease, the patients observe a gradual failure of their power of vision ; in others, blindness, more or less complete, sets in suddenly. I have heard a patient, upon coming to himself, after a uræmic convulsion followed by coma, ask to have the gas lit, although it was burning brightly at the time. This partial or total extinction of vision, too, used formerly to be referred to uræmic intoxication, and was called uræmic amblyopia or amaurosis. Latterly, however, the real source of this disorder has been found to be an extravasation into the retina, accompanied by inflammation. A diagnosis of Bright's disease has repeatedly been made by means of the ophthalmoscope alone. In one case which I have watched, the impairment and subsequent improvement of vision which occurred manifestly coincided with the formation and absorption of such extravasations.

[This disturbance of vision is not to be confounded with another —*uræmic amaurosis*—which appears sometimes superadded to an already existing retinitis, and sometimes comes on suddenly with the other uræmic signs, without previous derangement of the sight. In a few days, or even in a few hours, the sight fails so that a light cannot be distinguished ; but should the uræmia subside, vision may

soon be completely restored again. The speedy abatement of this form of blindness argues against its dependence upon permanent lesions. Somewhat more rarely, deafness and roaring in the ears have been noticed in uræmia.

Course.—The course of chronic diffuse nephritis varies greatly. In one class of cases the disease may last for ten or even for twenty years, and may be so latent that the patient is scarcely aware that he is ill, and so that a careless physician who neglects to examine the urine can make but little out of his patient's persistent but vague complaints of loss of strength and energy, of impaired appetite and digestion. When the inflammatory process runs this sluggish course, the urinary tubules take but little part in the changes which are going on in the interstitial connective tissue. There are but slight and merely transient inflammatory exudations into the tubules, whose epithelium suffers but little direct damage; hence, in such a case, but few if any casts or other characteristic forms are found in the urine. This languid process results merely in thickening of the renal connective tissue, followed by shrinking. It is this grade of inflammation, according to our view, which marks the distinction, both anatomically and clinically, between the forms of Bright's disease. Sometimes the disease maintains this insidious character throughout its course. The patient then preserves a tolerable appearance of health for years. His urine, which is copious and pale, shows but little albumen; but late in the disease he generally presents signs of a hypertrophy of the heart. In such a case death may take place suddenly in a uræmic convulsion, before there has been any dropsy whatever. In other cases attacks of eclampsia recur at irregular intervals. Still other patients die of apoplexy in consequence of the hypertrophy of the left heart and of the accompanying disease of the arterial walls.

In another and larger class the course of the disease is less sluggish and insidious. The inflammation is more active, and involves the tubules directly, so that dropsy soon develops. In some instances, which nevertheless are rare, the disease progresses steadily and with unabated activity to the end, causing the death of the patient in a few months by dropsy, secondary inflammations, or uræmia. Much more commonly there are marked fluctuations in its intensity. There will be periods, sometimes long ones, in which the urine retains its normal volume, is but moderately albuminous, and shows few casts or none at all. The dropsy abates and disappears; in a word, the symptoms of parenchymatous nephritis fade away or disappear. But between these intervals come exacerbations, during which, in spite of the most urgent and incessant efforts of the

patient to micturate, the daily secretion of urine is decreased, the percentage of albumen augments, numerous casts and cellular forms appear in the urine, and the dropsy makes further progress. During these long remissions of the malady the diagnosis may be difficult; or beyond a moderate albuminuria, which alone affords no proof of Bright's disease, the patient presents but vague symptoms, such as gastro-intestinal disturbance, anæmia, and loss of flesh. A patient is thus not unfrequently supposed to be convalescent, or even cured, until, sooner or later, relapses occur; so that, after an average duration of about two years, he succumbs to dropsy, to secondary inflammation of vital organs, or perhaps to uræmia. Should he escape all these dangers for a considerable time (as may happen exceptionally), then the active symptoms of the parenchymatous form of Bright's disease may give place to that of shrinking and atrophy. There will be a very profuse pale urine, of low specific gravity, containing a moderate quantity of albumen (sometimes, though very rarely, absent), and but few or no casts.

Prognosis.—While recovery from acute diffuse nephritis is by no means uncommon, the prognosis in the chronic form of the malady is far less favorable; and indeed it becomes almost absolutely bad as regards old or neglected cases, or when, from the condition of the heart and of the urine, an irreparable atrophy of the kidney can be made out. Nevertheless, even in some of these cases, though incurable, a hope may be held out of a tolerably comfortable existence for some years, or at least so long as the dropsy is moderate and transitory, as the serous cavities are free, and as the secondary lesions and the uræmia do not appear, and while the patient maintains his digestive powers. When the disease is recent and has been well and promptly treated, the prospects of complete cure are a little better. Sometimes the malady seems to run a medium course between that of the acute and chronic forms. The immediate danger is then much increased, but the hope that, after many weeks or even months, a complete recovery may take place, is much more warrantable than in the chronic form. In a diffuse nephritis which is secondary to some other grave disorder, such as an old malaria or a tedious suppuration, the nature of the primary affection must be considered in the prognosis.]

In respect to the duration and results of Bright's disease, it may be said that there are cases which run their entire course in a period of from six weeks to three months, and others in which the malady drags on for years. I used to know a physician of large practice in Altmark, who only died within a few years, although he had all the symptoms of Bright's disease as long as twenty years

ago. We have already alluded to the fluctuations in the intensity of the symptoms which take place in protracted cases. The most frequent termination of Bright's disease is death ; although patients more commonly die of the intercurrent inflammations than directly of the disease itself. Complete recovery may possibly take place, but it is extraordinarily rare. The longer the duration of the malady, so much the less is a favorable result to be looked for. In recent cases, the question always arises whether we have not to do with a croupous nephritis, which, as we have explained, admits of a better prognosis.

Treatment.—The causal indications require that a patient with parenchymatous nephritis should wear flannel next his skin, and exchange his dwelling, if damp and cold, for a dry and warm one ; and that he should be forbidden to go out at night or in bad weather, even during any temporary improvement. Well-to-do people, who dwell in bleak, damp, windy, seaside neighborhoods, should be induced to change their abode. Excesses in spirituous liquors, and the use of diuretics, cubebs, copaiba, and spices, are to be strictly prohibited. The discovery that Bright's disease is an inflammatory affection has done but little toward an efficient treatment of it. The so-called antiphlogistic method is inapplicable to any of its stages.

[The practice of blood-letting, the application of cold compresses, and the use of calomel no longer find advocates. We possess no means of extinguishing this obstinate and refractory disorder. Our task is therefore by no means simple, nor can any uniform plan of management be adopted for all cases. Its treatment must be chiefly a treatment of symptoms. The main disorder can only be attacked indirectly, by seeking to combat the individual pernicious or dangerous manifestations as they arise, and by striving to remove all influences tending to irritate the kidneys. From this point of view the scope and value of therapeutics in the varying phases of Bright's disease, as tending to prolong the life and to ameliorate the condition of the sufferer, should not be underrated.]

In fulfilment of the indications from the disease itself, derivation from the intestines by drastics, and from the skin by diaphoretics, has been proposed. We shall recur to the employment of this measure while treating of the management of the symptoms, as it often acts favorably upon the dropsy ; but we do not believe that the inflammation of the kidney can be arrested or allayed by such derivation. *Frerichs* speaks favorably of tannic acid, which he gives three times daily, in doses of from two to six grains, in combination

with aloes and in the form of a pill. The elimination of tannic acid through the urine in the form of gallic acid and pyrogallic acid, to which we have alluded elsewhere, warrants this treatment from a theoretical point of view. *Frerichs* himself admits that, although he has observed a diminution in the quantity of albumen excreted, yet, in chronic cases, he has but seldom seen a complete disappearance of it. My own experience does not speak at all in favor of tannic acid.

Bad as we have represented the prognosis of parenchymatous nephritis to be, we have often succeeded quite brilliantly in palliative treatment, and, in fulfilling the symptomatic indications, the therapeutics of Bright's disease are by no means to be regarded as powerless. We have recognized the loss of albumen from the blood as the immediate cause of most of the symptoms of the disease, and hence our most important task by far is to cover the loss of albumen by a diet rich in protein substances, and by appropriate medication. Soft-boiled eggs, milk, strong meat-broths, and roast-beef, in as large quantity as the patient is able to digest, are probably the best preventives of the dropsy; and, while patients in comfortable circumstances often withstand the loss of albumen with impunity for years, the poor, on the other hand, succumb to it far more speedily, because the former have better means than the latter to supply the daily losses which they suffer. Besides this, a moderate amount of beer or good wine should be prescribed, as by the use of these articles the waste of tissue is retarded and the nutrition promoted. Quinine and iron are the most suitable medicines. The former was thrown overboard by many physicians during the time of a generally prevailing nihilism in therapeutics, and its supposed tonic action upon the fibres has been much derided. But, in these days of reaction against such nihilism, tonics, and especially the preparations of quinia, have once more come into repute, and, indeed, it would seem that they do exert a beneficial action upon the state of nutrition by diminishing the consumption of material. Preparations of iron are equally appropriate, since their effect upon the formation of blood is most decided, and it is not the albumen of the blood alone but also the red blood-corpuscles which are reduced in quantity in this disease. Neglect of such directions as the above, a blind groping in search of a specific remedy, and a vague, planless exhibition of diuretics after the dropsy has set in, are merely so many tokens of incapacity on the part of the physician. In a series of cases, which have been described by Dr. Schmidt, in his inaugural thesis, I have obtained most brilliant results where all other treatment had failed, by putting the patients upon an exclusive diet of milk. The patients did not take a grain of any medicine whatever, but drank

from five to six pints of cows' milk daily. After the "cure" had been continued in this manner for about four weeks, some of the patients who, prior to the treatment, had been in the most wretched condition, had got rid of their dropsy, recovered an appearance of health, and regained so much of their strength as to be able to resume their business and even to perform hard labor. The albuminous character of the urine, however, has disappeared in but one case; in all the others it persisted. I am unable to account for the eminently beneficial action of milk upon Bright's disease. I propose to try whether it be possible to obtain a physiological explanation of these results by a careful analysis of the phenomena attending an exclusively milk diet, during health as well as disease, especially by careful weighing of the body and by taking accurate account of every thing taken into and ejected from the system.

If the above measures prove unsuccessful in averting the dropsy, or in allaying that which already exists, the establishment of active diaphoresis is strongly to be recommended. No benefit, however, is to be expected from the use of spiritus mindereri, the antimonials, and other so-called diaphoretics. I have known patients in an advanced stage of dropsy to rid themselves of it completely, in a few weeks, by the daily use of a hot bath, of a temperature of 80° to 100° F., followed by sweating for two hours in woollen blankets. The diaphoresis was so great in one case, that as much as 800 cubic centimetres of the sweat which had soaked through was collected in a wash-basin placed under the bed. All these patients were weighed before and after the sweating, and the clinical journals show that, during the sweating, they had lost two, three, and even four pounds in weight. However, it cannot be denied that, in some cases, this procedure also failed, nor that debilitated patients sometimes suffered so much from the process that I was obliged to desist from it. Finally, I may state that, in one instance, the abatement of the patient's dropsy was coincident with the first appearance of uræmic convulsions. The profuse drain of liquid from the skin naturally makes the blood more concentrated, and this accounts for the absorption of the interstitial effusion. Since, however, in parenchymatous nephritis, the effusion contains urea and perhaps other salts, it is readily conceivable that an active abstraction of liquid from the system would have the effect of overcharging the blood with these materials, and might thus give rise to uræmia. However, a closer investigation of the cases above mentioned has led to the conclusion that the supposed connection between diaphoretic treatment and the uræmic symptoms is improbable.

Whatever the theoretical objections against the employment of diuretics may be, yet, in desperate cases, recourse should always be

had to them. Squills and other stimulating diuretics must not be employed without the utmost caution, on account of the irritating action which they exert upon the kidneys; but there are certain salts, especially cream of tartar, and the tartarus boraxatus (soluble cream of tartar), which are decidedly beneficial in their effect. The physician above alluded to has repeatedly freed himself of his dropsy through the free use of buttermilk, and the employment of cream of tartar and small doses of Dover's powder.

We have yet to mention the drastics as remedies against the dropsy. Observations upon cholera have taught us that a heavy drain of water from the intestinal capillaries will render the blood more concentrated, and thereby promote absorption of dropsical collections. There was a very instructive case at the clinic of Tübingen, which has been described by *Liebermeister*, where a patient with Bright's disease was attacked by dysentery and died. In consequence of the thin, copious diarrhœa, the extensive general dropsy, from which this patient had long suffered, was reduced to a minimum a few days before his death. It may also be said in favor of the drastics that, during their employment, the kidneys are saved from irritation. Nevertheless, we should never have recourse to them save in time of extreme need, since the patient is liable to be intensely affected by them, and since, by their persistent use, the digestion becomes impaired. The drastics most frequently employed in treatment of Bright's disease are, elaterium, gr. $\frac{1}{6}$—$\frac{1}{2}$, colocynth, in form of decoction, ℥ j—℥ ij, to water, ℥ vj, or else in the form of tincture.

As remedies against uræmic intoxication, *Frerichs*, who ascribes this condition to surcharge of the blood with carbonate of ammonia, has proposed the acids, especially benzoic acid, in order to form harmless ammoniacal combinations. In the cases observed by me, I have not been able to perceive any effect from this treatment which is based purely upon theory; whereas strong drastics, and iced applications to the head, always seem to produce a favorable impression.

CHAPTER V.

NEPHRITIS VERA—INTERSTITIAL NEPHRITIS—RENAL ABSCESS—METASTATIC DEPOSITS IN THE KIDNEY.

Etiology.—While in acute and chronic Bright's disease the chief pathological changes take place in the uriniferous tubules, the alterations which occur in the intervening substance being altogether of a subordinate and secondary character, in the variety of renal inflammation which forms the subject of the present chapter, the disease lies

mainly in the scanty connective tissue which binds the tubules together. The most common causes of true nephritis are as follows:

1. Wounds and contusions. The kidney is seldom subjected to external violence, owing to its sheltered position. It is far more liable to injury from the presence of stones within the pelvis of the kidney.

2. Collections of decomposed ammoniacal urine in the pelvis of the kidney, the result of urethral stricture, enlargement of the prostate, palsy of the bladder from injury of the spine, and so forth. Here the irritation to which the kidney is subject is of a chemical instead of a mechanical nature.

3. Propagation of inflammation from the urinary passages to the kidney. It is easy to comprehend that an inflammation of the pelvis of the kidney, a pyelitis, might readily extend into the parenchyma of the organ, and cause nephritis; but the fact that nephritis is sometimes associated with gonorrhœa, but is not a result of extension of the latter disease by contiguity, and where there is no accumulation of urine in the pelvis of the kidney, is altogether enigmatical.

4. Propagation of the inflammation from the connective tissue of surrounding parts, the peritonæum and other organs. This is the rarest of all the modes of origin.

5. Embolism of small arteries of the kidney, and the introduction of septic or miasmatic material into the blood. This is the source of the so-called metastatic nephritis, observed in endocarditis, valvular disease of the heart, and in the various disorders classed under the general title of pyæmia, as well as in the infectious diseases.

There is usually no doubt as to the embolic origin of the cuneiform deposits, which, in the disease of the heart above alluded to, occur almost as frequently in the kidney as in the spleen; but it is sometimes extremely difficult to trace the origin of the small metastatic deposits which form in the kidney during the later stages of septicæmia, puerperal fever, typhus, etc., to the action of embolism. It is questionable whether interstitial nephritis ever arises from the effect of cold, or from that of acrid diuretics.

Anatomical Appearances.—In traumatic nephritis, or in nephritis arising from an extension of inflammation from the pelvis of the kidney, or from other organs, the kidney at first is enlarged, and is of a deep-red hue, which is either diffused over its whole substance, or else is confined to single spots in the cortical or medullary portion. Its consistence is much less firm. The albuginea is injected, thickened by infiltration, and easily detached. Upon section, the structure is indistinct, and the boundary between the cortical and pyramidal substance is effaced. A bloody, thick liquid can be expressed from the surface of the cut. At a more advanced stage the redness subsides.

The color of the renal substance becomes of a dirty brown or gray, owing to compression of its vessels by the interstitial exudation, which already, here and there, contains pus.

The discoloration usually begins at detached points, of the size of a hemp-seed. As it increases, these spots soften, until they finally break down into a purulent liquid. In this way small abscesses form, by the melting down of the renal substance, under the pressure of the constantly-accumulating pus-cells. In the cortical substance, the form of the abscess is more rounded; in the pyramidal, more elongated. The abscesses enlarge and coalesce, finally forming a great sac of matter, which may occupy one-half or even two-thirds of the kidney. Such an abscess may become incapsulated, and long remain embedded in condensed cellular tissue. In other cases, the deposits discharge in various directions, as into the pelvis of the kidney, the cavity of the abdomen, externally through long fistulous tracks, into neighboring parts of the intestine, which have become adherent to the wall of the abscess, or even, through the diaphragm, into an adherent portion of the lung. When the disease runs a more chronic course, it sometimes terminates differently. In such cases the interstitial substance of the kidney undergoes proliferation, while the peculiar tissues of the organ perish. At the close of such an attack of chronic interstitial nephritis, the kidney is irregular, and nodulated in shape. The elevations are more marked than those of the third stage of Bright's disease, and the albuginea is firmly adherent in the sulci between them. Upon cutting into one of these depressed spots, instead of renal-tissue proper, we find nothing except the indurated substance of a cicatrice.

The metastatic nephritis, which accompanies disease of the heart, shows no tendency to suppuration. In recent cases, distinctly circumscribed, dark-red cuneiform spots are found in the kidney. The base of the wedge lies toward the periphery of the organ, the apex toward its hilus. Microscopic examination shows an intense engorgement of the vessels with dark masses of blood, blood being also effused into and between the tubules. Hæmorrhagic infarction of the kidney, when of longer standing, undergoes metamorphosis similar to that already described as occurring in hæmorrhagic infarction of the spleen. A discoloration commences in the middle of the point of infarction, which, having undergone a complete fatty metamorphosis, and all the fat having been absorbed, cicatrizes, leaving a depressed scar. The metastatic deposits which form in the kidney in infectious disease, and in consequence of the absorption of putrid matter into the blood, are generally much smaller, and far more numerous, than the infarctions which occur in disease of the heart. Moreover, their tendency to break down is very great, so that, upon examination, it is not usual to find

solid spots, but merely abscesses in the kidney, surrounded by an area of redness.

Symptoms and Course.—When not of metastatic origin, acute interstitial nephritis, like acute inflammation of other important organs, sometimes commences with a rigor. At the same time there are violent pains in the region of the kidney, and, in this form of renal inflammation, there is a pain, which is hardly ever absent, which becomes almost intolerable upon the most moderate pressure, and shoots along the ureters to the bladder, and toward the testicle and thigh of the affected side. The sympathetic vomiting, which we have described as an almost constant accompaniment of acute parenchymatous nephritis, is nearly always present also in this form of nephritis. Owing to compression of the urinary tubules and Malpighian capsules by the interstitial exudation, the secretion of urine is repressed. That which is passed is concentrated, dark, and is often mingled with blood. The fever which accompanies this disease from the outset is very apt to assume a typhoid character. The patients become disturbed in mind, delirious, somnolent, and fall into a state of stupor, with convulsions; symptoms which are to be ascribed to a suppression of the urine, and to a surcharge of the blood with excrementitial matter. The disease may terminate fatally, in the course of a few days, through general paralysis of the nervous system.

The occurrence of suppuration in the kidney may be suspected when the disease continues without remission, and when its course is marked by numerous chills, and, above all, when pus is discharged with the urine. When an abscess forms in one part of the kidney, the rest of which has not been affected, or has regained its healthy condition, the disease takes a more chronic course; but the fever continues, and consumes the patient gradually, who almost always dies sooner or later of a phthisis renalis. To detail the various modifications of symptoms which arise from the complications of pyelitis, cystitis, and the bursting of renal abscesses in various directions, would lead us too far. The different courses which the matter may take have already been mentioned above. There is a form of interstitial nephritis, which is very difficult of recognition, in which the substance of the kidney gradually perishes, and is replaced by a new growth of connective tissue. A permanent derangement of the urinary secretion, a constant desire to pass water, dull pains in the region of the kidney, dropsy, great listlessness, which gradually increases to coma, with other indications of the so-called uræmic poisoning, are the symptoms of this form of disease, but they are very rarely properly interpreted.

Metastatic interstitial nephritis, not having any characteristic symp

toms, is usually overlooked during life. However, I have sometimes noticed that the formation of a large hæmorrhagic infarction in the kidney is accompanied by a chill, that the patient complains of severe pain in the kidney affected, and that the urine is scanty and contains blood. When we encounter symptoms like these, in a case of disease of the heart, we may confidently diagnosticate the existence of an infarction of the kidney, particularly when there is or has been evidence of embolism elsewhere. Renal metastases, which form during septicæmia, puerperal fever, etc., are usually mere "accidental" discoveries upon the dissecting-table. We cannot even suspect their existence during life.

Treatment.—As, in most cases of interstitial nephritis, it is impossible to meet the causal indication when the affection proceeds from the presence of a stone, or from a collection of putrid urine in the pelvis of the kidney, or from embolism or infectious disease, we must confine our efforts to an antiphlogistic treatment, which is much more appropriate in this case than in those heretofore described. The repeated application of leeches, cut cups, and of cold over the kidney, with the subsequent use of long-continued warm baths and warm poultices, and the exhibition of drinks containing carbonic acid, are the most approved measures. The sequelæ, especially the abscesses and fistulæ which may form, must be treated according to the symptoms.

CHAPTER VI.

PERINEPHRITIS.

Etiology.—In rare instances the adipose tissue, in which the kidney is enclosed, becomes the seat of a primary, independent inflammation, which is usually the result of an injury, or, as has been claimed now and then, of exposure to cold. Much more frequently the inflammation is secondary to a suppurative action within the pelvis or substance of the kidney. I have seen one case in which a pericystitis extended along the ureter to the fat around the kidney.

Anatomical Appearances.—As a rule, inflammation of the areolar envelope of the kidney soon results in suppuration. The tissues then become discolored, and their meshes are filled with pus. The small collections of pus coalesce and an abscess forms, often of very considerable magnitude, and which may point in almost any direction. In other cases there is no suppuration, but the loose cellular tissue becomes condensed and indurated, and is converted into a thick fibrous rind.

Symptoms and Course.—When perinephritis is acute, its symp-

toms bear a strong similarity to those of acute interstitial nephritis. A violent fever, which sometimes is ushered in by one or more rigors, and by severe pain in the region of the kidney, which becomes intolerable whenever the adjacent muscles of the body contract, or are subjected to any strain by movement of the body, is a symptom common to both diseases. There is one important distinctive point between the two; in pure uncomplicated perinephritis, there is no suppression of urine, nor does the urine contain either blood, albumen, or pus. If the disease goes on so as to give rise to a large abscess, a tumor appears in the renal region, which fluctuates with greater or less distinctness. If the abscess break into the cavity of the abdomen, it occasions an acute peritonitis, which speedily terminates in death. Recovery may take place where the abscess discharges into the intestine, or where it points externally, or is opened artificially. External opening of the abscess generally occurs in the back, below the false ribs, and is usually preceded by an excessive aggravation of the pain upon movement of the body, and by a more or less extensive œdema of the skin over the region affected. In other cases the pus descends along the psoas muscle, giving rise to a symptomatic abscess, which usually makes its appearance below Poupart's ligament.

Treatment.—The most appropriate treatment of a recent case of perinephritis is local blood-letting, and, in a later stage of the disease, the systematic application of cataplasms and, the use of lukewarm baths. Abscesses must be opened as soon as possible, according to surgical rules, and should be kept open for a while.

CHAPTER VII.

AMYLOID DEGENERATION OF THE KIDNEY—PARENCHYMATOUS NEPHRITIS, WITH AMYLOID DEGENERATION.

The kidney, like the liver and the spleen, not unfrequently undergoes a degeneration, by the deposit in the elements of its tissues of a material whose reaction against iodine and sulphuric acid resembles that of the cellulose of plants, but whose chemical constitution is more like that of the protein substances. Amyloid degeneration of the kidney takes place under conditions similar to those under which it occurs in the liver and spleen, its causes being severe chronic disease, such as syphilis, mercurial poisoning, rachitis, consumption of the lungs, and long-standing suppuration, such as occurs in caries and necrosis.

The degeneration probably always commences in the walls of the blood-vessels, particularly in those of the glomeruli, to which it usually

remains limited; while, at the same time, the epithelium of the tubules undergoes the changes already described in Chapter IV. The name "parenchymatous nephritis, with amyloid degeneration," is, therefore, more appropriate for this form of disease than the term "amyloid or lardaceous degeneration of the kidney." Even microscopic examination is not by itself sufficient to enable us to distinguish between the affection in question and simple parenchymatous nephritis. But, if a thin slice of the specimen be treated for a few moments with a solution of iodine, the red color of the glomeruli becomes so distinct even to the naked eye, that, even before resorting to the microscope, the appearance of a multitude of small red dots, standing out in contrast to the surrounding yellow ground, makes the diagnosis almost certain. Under the microscope the loops of the glomeruli seem remarkably large, and present a peculiar dead lustre. The Malpighian capsules, too, often exhibit a broad, homogeneous, dull outline. If, prior to the examination, the preparation have been laid for a time in a dilute solution of iodine, the bodies above described will assume the characteristic yellowish-red color. Upon the subsequent addition of a few drops of sulphuric acid, they become of an indistinct violet or dark blue. It is very unusual for the tunica propria, and still more so for the epithelium of the urinary tubules, to participate in the amyloid degeneration.

When an individual, who hitherto has enjoyed good health, is affected by albuminuria, dropsy, and deterioration of the blood, it is so very improbable that he is suffering from amyloid degeneration of the kidney, that that disease may confidently be excluded from the diagnosis. On the other hand, the appearance of similar symptoms in a person who has long been afflicted by syphilis, consumption, tedious suppuration, or any other exhausting malady, makes it extremely probable that we have to deal with an amyloid degeneration of the kidney, or, more properly speaking, with a parenchymatous nephritis with amyloid degeneration of the walls of the renal vessels. If the patient also have an enlargement of the liver and spleen, and if the portions of these organs accessible to palpation present the characteristic resistance of amyloid degeneration, the diagnosis is still more sure. In distinguishing between amyloid degeneration and simple parenchymatous nephritis, *Traube* lays great stress upon the high specific gravity and dark color of the urine in the former disease. My own observations fully confirm the truth of *Traube's* views, and I may add that I have been struck, not only by the darkness of the urine, in amyloid renal degeneration, but also by its unnatural, yellowish-brown color; moreover, my colleague, *Hoppe-Seiler*, has shown that such urine contains extraordinary quantities of indican. There is nothing in the quality of the exudation-casts, or in the degree of frequence of uræmic

symptoms, whereon to base a differential diagnosis between a simple parenchymatous nephritis and one which is complicated by amyloid degeneration. Moreover, the difference between the two conditions is a matter of but little practical interest.

The treatment recommended for amyloid degeneration of the liver and spleen is equally appropriate in amyloid disease of the kidney. It is questionable whether a retrogression of the disease be possible. The preparations of iron and iodide of iron may act beneficially upon the primary disease, but can hardly cure a degeneration of the kidney.

CHAPTER VIII.

GRANULAR DEGENERATION OF THE RENAL EPITHELIUM.

[Frequently on autopsy we find the kidneys flabby, bloodless, of a pale, dirty, grayish-red color, somewhat enlarged, or of normal size. From their cut surface we may scrape off a cloudy grayish pulp. This pulp consists of epithelial cells swollen by albuminous infiltration and dotted with numerous fine granules, which render the nuclei indistinct, but which vanish in the presence of potash and acetic acid (thus showing that they consist of albuminous particles). At a later period fat-granules may appear. When the disorder is intense, the kidney becomes tender and flabby, and the epithelium falls off ; but, if of only a moderate degree, the cells may undoubtedly be restored to their normal condition. Proliferation of the epithelium occurs, but very rarely, and then only late in the disease. Granular degeneration is not peculiar to the kidney, but is often found simultaneously in the cells and tissues of the most diverse organs, particularly in the liver, the heart, and the muscles. This widespread appearance of the disease shows that its cause must be a general one. In the bodies of persons who have died of infectious diseases—pyæmia, typhus, or acute miliary tuberculosis—more or less marked granular degeneration is found, as a result either of some abnormity of the blood, or perhaps as a consequence of the persistent elevation of the temperature which attends these diseases, and which, according to *Schultze*, exercises a most pernicious influence upon cellular formations. But the disorder also is found when there has been no fever, in chronic anæmia, chronic disease of the heart or lungs, and in poisoning by phosphorus, sulphuric acid, and carbonic oxide. Sometimes the cause of the granular degeneration is purely local, and is limited to the kidney where it is found, supplementing other morbid conditions. Very important

results are ascribed to the more intense grades of this lesion. Thus it is supposed in acute forms often fatally to cripple the action of the heart.

The condition is also found in the bodies of pregnant and puerperal women. At present we have no definite knowledge as to how pregnancy affects the minute structure of the kidney and other organs in these cases.]

The most common and frequently the sole symptom of parenchymatous degeneration of the kidney is the presence of albumen in the urine. We may say, too, that whenever there has been albuminuria during pregnancy or during grave disease, the renal epithelium will be found to be in a more or less advanced state of degeneration after death. How often the question is asked by the anatomist during the autopsy, whether the patient had albuminous urine! On the other hand, I will not venture to state positively that the albuminuria, which so often accompanies the conditions above referred to, is always the result of degeneration of the epithelium of the kidney, or that degeneration of the renal epithelium is always attended by albuminuria. The quantity of albumen which the urine contains is probably never so great in parenchymatous degeneration as in amyloid degeneration, and parenchymatous inflammation of the kidney. When it arises after acute febrile disease, it never produces dropsy. This symptom is often absent, too, during pregnancy accompanied by parenchymatous renal degeneration and albuminuria. Sometimes, however, there is a moderate degree of dropsy, and in rare instances a very large one. When it accompanies pregnancy, it is sometimes a source of danger, owing to the eclamptic attacks which it provokes during and immediately after parturition, and of which we shall have more to say when we come to treat of diseases of the nerves.

If the original disease terminate in recovery, the nutritive disorders of the renal epithelium to which it has given rise are also completely repaired. Soon after delivery the albuminuria and dropsy disappear in almost all cases, another proof that the malady in question is not to be confounded with interstitial nephritis. We are quite ready to admit that in most cases of eclampsia puerperarum there is renal disease; but we deem it quite inadmissible to attribute the albuminuria, dropsy, and eclampsia to "Bright's disease."

After what has been said already regarding parenchymatous degeneration, further discussion of its treatment becomes superfluous. We shall treat hereafter of the proper management of the eclampsia.

CHAPTER IX.

CARCINOMA OF THE KIDNEY.

Of the malignant neoplastic growths, carcinoma is the one most frequently seen in the kidney. Renal cancer is sometimes primary; sometimes it accompanies carcinoma of other organs, as a secondary formation. Young persons, and even children, are quite frequently attacked, although it is somewhat more common in advanced age.

Cancer of the kidney generally assumes the medullary form. Scirrhus and colloid are far more rare. The former sometimes develops in the form of circumscribed nodules of varying size, which gradually replace the parenchyma of the kidney. Sometimes the cancerous degeneration spreads farther and farther into the surrounding tissues from its original point of development, so that the kidney gradually becomes transformed into cancer (*Rokitansky*, infiltrated cancer). A carcinomatous kidney may attain an enormous bulk sometimes, forming a nodular tumor of the size of a child's head. The degeneration often involves the lymphatic glands in the hilus of the kidney, whence it spreads to the retroperitoneal and mesenteric lymphatics, and to the ligaments and periosteum of the spinal column; or else it may grow inward into the cavity of the organ, and thence into that of the adjoining veins. The frequency with which cancer of the kidney is complicated with cancer of the testis is a matter of importance, which calls to mind the still more common coincidence of tuberculosis of these organs. Hæmorrhages both within the tumor and on its periphery are very apt to occur. In the latter case, the blood is effused either into the peritonæum or else into the urinary passages.

The disease often remains latent for a long time. It is usually by the gradual advance of a marasmus, for which no other cause can be assigned, that suspicion is awakened of the existence of a malignant tumor in a region inaccessible to palpation. There may be no pain at all in the lumbar region, and, when it does exist, it is not characteristic. The renal secretion may go on undisturbed, and the urine may be quite free of blood and albumen. As the disease advances, the tumor formed by the cancerous kidney, which is often of enormous size, can generally be felt through the abdominal walls, especially when the latter have become wasted and relaxed. The form of the tumor, and especially its immobility, will prevent our mistaking the enlarged kidney for an enlargement of the liver or spleen. It cannot be moved from side to side, nor does it follow the motions of the diaphragm. Very large cancers of the right kidney sometimes produce a remarkable and peculiar displacement of the liver inward, causing it to turn upon its

long axis, so as to bring a large part of its convex surface in contact with the abdominal wall. Hæmaturia and albuminuria are absent about as frequently as they occur. As already stated, part of the bleeding proceeds from the vessels of the growths which push into the urinary passages, while another part is the result of excessive hyperæmia of the surrounding tissues. Owing to the large quantity of blood thus discharged, the hæmaturia often forms one of the most prominent symptoms of this disease. The complexion of the patient assumes the dirty hue so common in cancer, and he dies of exhaustion which progresses all the more rapidly when the hæmorrhages are frequent and profuse; unless, indeed, death ensue in consequence of some intercurrent disorder, or from the invasion of other vital organs by secondary cancer. Treatment is ineffectual, and must be limited to husbanding the patient's strength, repression of the hæmorrhages, and removal of coagula from the bladder, according to surgical rules.

CHAPTER X.

TUBERCULOSIS OF THE KIDNEY.

Rokitansky recognizes two forms of renal tubercle. The first form is symptomatic of a tuberculosis involving several or even the majority of the organs. In acute miliary tuberculosis, gray nodules, similar to those found studding the lungs, pleura, peritonæum, etc., are also found in the albuginea and parenchyma of the kidney. In extensive chronic tuberculosis, tolerably large, yellow, cheesy deposits of tubercle are sometimes met with in the kidneys, but they rarely contain collections of softened tubercular matter or tubercular cavities. The first of these forms has but little effect upon the action of the organ. It is unrecognizable during life, and is of more moment in a pathological than in a clinical point of view.

The second form of renal tubercle is generally complicated with tuberculosis of the testicle, prostate, seminal vesicles, and urinary passages. It is not constantly preceded by pulmonary tubercle; but this disease almost always sets in at a more advanced period of renal disorder. In this second form the deposit is very copious, and the individual nodules soon coalesce into large tubercular masses. The organ becomes enlarged, assumes an irregular, knobby shape, and within it we find large cheesy collections, some of which are filled with tuberculous pus. This form of renal tuberculosis, too, would likewise frequently escape observation, were not the diagnosis almost always aided by the complications above mentioned. The longer a chronic disease of the urinary passages has coexisted with the admixture of pus, and now and

then of blood in the urine, the tuberculous character of which is indicated by the coexistence of an enlargement of the testicle, so much the more likely will it be that the kidney itself has become involved in the disease. The diagnosis receives further confirmation if we are able to feel an uneven tumor in the region of the kidney, through the flaccid walls of the abdomen.

CHAPTER XI.

PARASITES IN THE KIDNEY.

The echinococcus is the parasite most frequently found in the kidney, although even there it is met with less frequently than in the liver. Its presence in this organ is originally due to the existence of the embryo of the tenia echinococcus in the intestinal canal (see Vol. I.). We have no knowledge as to why the young brood, in their emigration from the bowel into the other organs of the body, should sometimes enter the kidney. The echinococcus sacs, which occasionally attain the size of a fist or of a child's head, are quite like those found in the liver and spleen. They are embedded in a fibrous capsule belonging to the kidney. They may atrophy and burst, discharging their contents in different directions. They may also occasion inflammation and suppuration in the parts about them, and the renal abscess thus resulting may burst into the peritonæum, intestine, or pelvis of the kidney.

The development of echinococci in the kidney may be entirely unattended by symptoms. In some instances, however, the patients complain of a dull pain in the lumbar region, which we cannot well account for. A tolerably sure diagnosis may be made out if we can feel an irregular nodular tumor in the region of the kidney, and at the same time can exclude carcinoma, tuberculosis, and hydronephrosis, to be treated of hereafter. Certainty is only possible when cysts of the echinococci or traces of them are discharged with the urine. The hyaline walls of the daughter cysts, consisting of concentric layers, distinctly recognizable under the microscope, are not easily mistaken. Symptoms of renal colic may arise during their passage through the ureters, and their discharge from the bladder is often attended by the utmost distress, especially in men.

[The strongylus gigas is the largest of known round worms. The female may attain the length of half a yard or more. Its mouth is bordered by six wart-like projections. The male is smaller, and at his tail-end has a cup-shaped appendix, at the bottom of which is the genital opening with the penis protruding from it like a hair. The method of development of this parasite has not been ascertained.

Schneider found worms in certain fish which seemed to represent the early forms of a strongylus; and the fact that the strongylus species most generally infest fish-eating animals argues in favor of this view. Its occurrence in man is very rare indeed. According to *Davaine*, but seven well-authenticated cases have been collected. We know, however, that in brutes the creature usually occupies the pelvis of the kidney, more rarely descending the ureter into the bladder, and that its presence causes symptoms similar to those of calculus, and may cause retention of urine and hydronephrosis.]

CHAPTER XII.

DEFORMITIES OF THE KIDNEY—IRREGULARITIES OF ITS SHAPE AND POSITION.

Absence of one kidney usually is accompanied by abnormal magnitude of the other, and the secretion of urine remains normal. Union of the two kidneys, which then are generally connected by a narrow bridge of renal substance at their lower ends—the so-called horseshoe kidney—is a matter of mere anatomical interest, and of no clinical importance. The same is true also of lobulation of the kidney, which depends upon a persistence after birth of the fœtal condition of the organ, and is distinguishable from an acquired lobular state of the kidney by the healthy condition of the renal parenchyma and capsule at the depressed points. Irregularity of position is most common in cases of horseshoe kidney; the united organs then usually lie much deeper than natural, sometimes as low down as the last lumbar vertebræ. But, even without the coexistence of this malformation, an unnaturally deep position of the kidneys is not an uncommon congenital deformity, and is usually accompanied by irregularity in the origin and number of the renal vessels, as well as by anomaly in the lengths of the ureters.

Misplacement of the kidney, with abnormal mobility, is a matter of greater importance. Quite a considerable number of cases of this anomaly have been observed since attention was first called to it. In Greifswald alone I know of three cases of movable kidney. The kidney (and almost always the right one) lies embedded in a loose areolar tissue. Its vessels are elongated, and have a sort of mesentery formed out of the reduplication of their peritoneal coat. Such anomalies are most common among women whose abdominal integuments have become greatly relaxed by repeated child-bearing. According to *Rayer*, it may also result from violent concussion of

the body—as from a fall from a great height. In other cases there is no other anomaly, nor is there any apparent cause for the mobility of the viscus. When the patient stands erect, the movable kidney may be felt usually below the liver, or even still deeper. Its characteristic bean shape is distinctly recognizable, and it often can be pushed a considerable distance to the right or left, but more easily upward and downward. A patient in my ward, by moving and shaking his body, was able to get his kidney into a great variety of positions. There are either no evil results whatever attending the affection, or they depend upon complications, although colics and slight inflammations of the peritonæum may result from pressure of the movable organ upon the other viscera. The knowledge that she has a tumor in her abdomen often acts very injuriously upon the spirits of the patient, who sometimes becomes hypochondriac. Little can be done by way of treatment for a movable kidney ; but the patient (particularly if she have very flabby abdominal walls) generally feels better when she wears an elastic bandage of gum elastic or knit cotton.

CHAPTER XIII.

ADDISON'S DISEASE—BRONZED SKIN.

[This disease, first described by *Addison*, still presents an obscure problem, notwithstanding the laborious researches of many of our most acute observers (*Virchow*, *Meissner*, *Averbeck*, *Klebs*, *Eulenburg*, *Guttmann*, *Risel*, and others). All former doubts as to the real existence of such an independent affection are now dissipated ; but it has been shown that the lesions which used to be thought fundamental and characteristic of it, namely, the morbid state of the suprarenal capsules and the dark stain upon the skin, though commonly present, are not its essential features. The original assumption that Addison's disease depends upon a degeneration and destruction of the suprarenal capsules is contradicted by the negative results of experiments made in seeking out the hitherto undiscovered functions of these organs, as well as by the fact that the capsules have been found destroyed or in a state of disease in bodies of persons who during life presented none of the symptoms of the malady. Attention was thus directed to the sympathetic plexus of nerves adjacent to the capsules, the implication of which in this affection *Addison* himself had already pointed out ; and the opinion was gradually reached by a number of English, French, and German observers that a morbid state of the abdominal sympathetic, par-

ticularly of the cœliac plexus and semilunar ganglia, and of the nerves connecting them with the suprarenal capsules, is of essential importance in the disease. The circumstance which speaks most in favor of this view is, that in most of the cases in which the sympathetic has been examined, besides the suprarenal lesion usually present, there has been also found either redness, swelling, thickening, fatty degeneration, atrophy, pigmentation, or wasting of the ganglia. But out of twenty-nine cases collected by *Guttmann* and *Eulenburg* the sympathetic was found more or less altered in nineteen and normal in ten. This negative result, though of only a minority of cases, reduces the above attempt at explanation to a mere theory; the more so when we read that experiments by *Adrian Schmidt*, *Lamansky*, *Schiff*, and others upon the abdominal ganglia give rise to no symptoms suggestive of Addison's disease. A further hypothesis was then advanced, that the disorder depended upon derangement of the central nervous system and not upon that of the sympathetic; and *Rossbach* describes it as a hitherto undefinable functional disturbance of the nervous system lying in close relation to, but not inseparable from, the suprarenal capsules, and characterized by psychic disorder, intense anæmia, debility, and very often by a dark staining of the skin.

In order to account for a connection between a nervous disturbance and a morbid stain upon the skin, the cases of albinism have been invoked in which the anomaly is very distinctly limited to a district of skin corresponding to a certain nerve-district, as have also the cases of sudden turning gray of the hair under the influence of mental emotion. The deposits of pigment which form in the skin of pregnant women, and in many who suffer from menstrual and uterine disease, have also been pointed out in support of the view that staining of the skin may proceed from disorder of the suprarenal capsules.

The anatomical changes of the suprarenal capsules found in Addison's disease are not always the same. In most cases there is a caseous fibrous degeneration resulting from chronic inflammation of the organ. The capsules are often of two or three times their normal size, sometimes even as big as a hen's egg, and are converted into masses of dense connective tissue containing cheesy or calcareous nodules. Their surface is irregular and knotty, and often adherent to adjacent structures. Their consistence is tough and hard. The inflammation, instead of being general, may however be only partial and limited to single spots. The disease is generally bilateral.

The many cases in which both capsules have been found totally destroyed argue against the theory that the suprarenal capsules are

secreting organs, and Addison's disease a result of over-production of deleterious chemical matters in the blood (particularly taurocholic acid), which partially dissolves the blood, thus causing the anæmia, while a precipitation of the pigment of the blood occasions the bronze color. In rarer cases, instead of this degeneration, we have found carcinoma, extravasation of blood in the tissues of the capsules, and sometimes simple atrophy ; hence it must be concluded that the bronze color is not due to any special process. The immediate cause of the brown skin is a deposit of pigment in the rete Malpighii ; but pigment-cells are also to be found in the true skin, and are to be distinguished from those in the rete Malpighii by their containing iron.

There are sometimes no lesions whatever of other important organs ; but more frequently there will be found enlargement of the spleen and of the intestinal follicles, cheesy degeneration of the mesenteric glands, and sometimes caries of the vertebræ.]

Symptoms and Course.—The darkening of the skin, whence the malady derives one of its names, although it is by no means its most important or most dangerous symptom, occurs gradually. If the patient die before it has become very pronounced, it is apt to be overlooked entirely. Such cases, however, do not disprove the connection between disease of the suprarenal capsules and the deposit of pigment in the rete Malpighii. It is only by degrees that the discoloration of the linea alba and nipples in pregnancy acquires a sufficient degree of intensity to become conspicuous, and indeed there are many persons in whom it is never very marked even at term ; and yet no one denies the existence of a connection between pregnancy and such pigmentary deposit.

In some instances the skin becomes so dark as to resemble that of a mulatto or negro. One of my patients, a baker, from Heilbronn, used to be generally known as "Black R——." The color sometimes is a pure gray, inclining to black, like plumbago. At other times it has a brown or yellowish tinge. It generally commences upon the exposed parts of the body, but, although more intense upon those regions, is by no means confined to them, a circumstance which might save us from errors of diagnosis in dealing with sailors, field laborers, and others whose hands and faces are often of an exceedingly dark grayish-brown tinge. The roots of the nails also remain white. The palms of the hands and soles of the feet are spotted here and there, but are not uniformly discolored. The sclerotica never participates in the discoloration, and its pearly hue often forms a marked contrast with the dark color of the face. Black spots upon the lips and mouth seem to me to be pathogno-

monic of the affection. At all events, no mention is made of them in any of the negative reports of bronzed skin without disease of the capsules.

Its more serious symptoms consist in an extreme debility, often combined with deep depression of spirits. In some instances this debility increases from time to time into profound and long-continued swoons. Sometimes the adynamic symptoms are so prominent as to remind one of a severe attack of typhus, or of some other acute affection, with so-called typhoid symptoms. Mistakes for typhus, however, may always be avoided if all the elements of the case be attentively considered, especially if the temperature be observed. If, however, the case has not been long under observation, and if the discoloration of the surface be not very distinct, it may be impossible to make a positive diagnosis. The great lassitude and insurmountable sense of weakness, unattended by any apparent cause of exhaustion, would seem to proceed from a disorder of innervation. The richness of the suprarenal capsules in nervous elements, and the numerous communications existing between them and the various nervous plexus, especially the solar plexus, rather confirm such a supposition.

Pain in the back, and still more frequently pain in the epigastrium, is a very frequent but not a constant symptom. In one of my cases the pain was so violent that the patient for weeks was treated with poultices. In a second, severe pain in the epigastric region was one of the most conspicuous symptoms, while in the third the patient never complained of pain at all.

Dyspepsia and vomiting are reported as occurring in nearly every case ; yet even these symptoms are sometimes absent. The vomiting of Addison's disease may be regarded as a so-called sympathetic vomiting ; since, in disease of other organs adjacent to the stomach, and even in such as contain fewer nerves than the suprarenal capsules, and whose nervous connection with the stomach is much less obvious, such sympathetic vomiting occurs.

Diarrhœa has been observed in many cases. In two of my three cases the dirrahœa was very obstinate. It would be easy to account for this diarrhœa, could it be proved *post mortem* that the cœliac ganglion always was diseased ; since, according to the experiments of *Budge*, all the rabbits whose cœliac ganglia he removed suffered from diarrhœa. But independently of this, there is nothing surprising in the occurrence of diarrhœa in disease of the suprarenal capsules, these organs being richly endowed with nerves, and standing in intimate relation with the nervous plexus of the abdominal

organs, and since, too, there is no doubt as to the dependence of liquid discharges upon disturbance of innervation.

The convulsions and other grave nervous disorders which sometimes occur are difficult to account for. One of my patients had repeated epileptic attacks. *Post-mortem* examination usually affords no clew as to the origin of these symptoms.

The temperature of the body is not increased, unless there be some complication of a febrile character.

The beat of the heart and the pulse is accelerated, and toward the close of the malady is often extremely feeble. The great frequence of the pulse stands in striking contrast with the lowness of the temperature, and, if no note be taken of the latter, it is liable to cause an erroneous impression that there is fever.

The progress of the disease is almost always chronic. There are but two or three known cases where the process has run an acute course, speedily terminating in death.

Although there are a few cases in which a temporary improvement has been observed, yet we have no well-authenticated instance of complete recovery ; and death must be regarded as the most frequent, and probably, indeed, as the sole termination of this malady.

Treatment.—Of course, the treatment of Addison's disease must be a mere treatment of symptoms. In one of my cases it was very evident that good nourishment and careful nursing were not without a beneficial effect upon the course of the disorder. The patient, a poor servant, was repeatedly received at the clinic, in a state of extreme emaciation, and unfit for any labor. He always gained considerably in weight in the course of a few weeks, and so far recovered his strength that he could be employed in a variety of occupations ; until, three years and a half after his first reception at my clinic, he suddenly and unexpectedly died in one of the above-mentioned epileptiform attacks.

SECTION II.

DISEASES OF THE PELVIS OF THE KIDNEYS AND URETERS.

CHAPTER I.

DILATATION OF THE PELVIS OF THE KIDNEY, WITH ATROPHY OF THE RENAL SUBSTANCE—HYDRONEPHROSIS.

ETIOLOGY.—Whenever the flow of the urine, through the ureters, into the bladder, becomes permanently obstructed, the urine accumulates in the pelvis and calyces of the kidney. If the pressure of the urine within the pelvis of the kidney be very great, the openings in the tubuli recti and papillæ are closed, and the papillæ themselves become compressed. The urine then ceases to flow from the kidney into its pelvis, and distention of the latter would progress no farther were it not that the pelvis and calyces possess a mucous membrane, and still continue to produce mucus, which is mingled with the accumulated urine. Hence more and more liquid collects in the renal pelvis, distending the latter, till finally the pressure flattens out the papillæ, and induces a gradual atrophy, which may terminate in the total loss of the substance of the organ.

Closure of the ureter may proceed from the impaction into it of a stone. Far more frequently, hydronephrosis depends upon compression of the ureter by a tumor, or by a callous contracting cicatrix in the peritonæum which covers it, or by a growth, especially by a cancer, of the subperitoneal connective tissue. In other cases, closure of the ureter is the result of inflammation, which has induced swelling of its mucous membrane, or adhesion of its walls. The lower and the closer to the bladder the obstruction is, so much the larger is the portion of ureter which takes part in the dilatation.

Moderate degrees of dilatation of the uriniferous apparatus sometimes arise from an impediment to the discharge of urine, which exists

in the urethra or bladder, but, in such cases, the affection being bilateral, the dilatation of the renal pelvis can never become so great as to close the mouths of papillæ of the kidneys without involving danger to life. It seems strange that, on autopsy, hydronephrosis of both sides, which seems to have existed a long time, is frequently found.

Anatomical Appearances.—According to *Rokitansky*, in the milder degrees of hydronephrosis, the papillæ are shrunken, hardened, and leathery. Gradually the papillæ disappear in the concavity of the dilated calyx, while above it the renal substance diminishes, its tissues becoming compressed, leather-like, and tough. In extreme cases, the substance of the kidney is only a line or two in thickness, finally disappearing, leaving a membranous sac, of a lobulated appearance externally, and cellular within. The cells communicate with each other, their walls having burst or atrophied; they contain a urinous liquid, clear and serous, or turbid and containing a sediment, varying greatly in character. These sacs may attain the size of a child's head, or even that of a man's. A dilated ureter may attain almost any conceivable size, its calibre equalling or even exceeding that of the small intestine. The walls of the ureter are thickened, and the tube itself, instead of running straight to the bladder, is tortuous and convoluted, like a gut.

Symptoms and Course.—Hydronephrosis cannot be detected, except in its most intense form, and, of course, this cannot exist when both ureters are obstructed. The secretion of urine is not diminished, as the active kidney acts vicariously for the disabled one. There may not be any pain in the lumbar region. Diagnosis is solely based upon the discovery of a somewhat soft and indistinctly-fluctuating tumor in the region of the kidney. If there be any temporary abatement of the obstruction to the escape of urine from the organ, the tumor may diminish from time to time, and such an occurrence sometimes aids the diagnosis. When the sac becomes inflamed (as has sometimes been observed), there is much pain, accompanied by severe rigors. The pus which forms may point in various directions, thus giving great diversity to the character of the symptoms. Should the impediment which has obstructed one ureter extend so as to prevent the flow of urine into the bladder from both kidneys, all secretion of urine will cease, and the patient will speedily perish, under symptoms of uræmia.

Treatment.—If the obstruction lie in the urethra only, diligent catheterization may prevent dilatation of the urinary passages, or arrest such dilatation when it has commenced. It is out of our power, however, to arrest or to allay the graver forms of dilatation with atrophy of the renal substance, which proceed from obstruction of a ureter unless the obstruction of the ureter be caused by a tumor, susceptible of relief by medical or surgical interference.

CHAPTER II.

INFLAMMATION OF THE PELVIS OF THE KIDNEY—PYELITIS.

Etiology.—In rare cases, the pelvis of the kidney is the seat of a croupous or of a diphtheritic inflammation. This is usually accompanied by similar inflammation of other mucous membranes, and is usually a concomitant of infectious disease. It is most commonly observed in the typhoid stage of cholera, but may also proceed from catarrhal inflammation, especially that arising from calculous pyelitis.

Catarrhal pyelitis, in the great majority of cases, is the result of an irritation of the mucous membrane, induced by the presence of calculi in the pelvis of the kidney. In similar manner, the ammonia of stagnant and putrid urine may provoke a most intense catarrh of this region. Much more rarely, catarrh of the urinary passages is caused by acrid substances, such as cantharides, the balsams and resins, which have been taken into the system, and excreted with the urine. Again, we frequently find pyelitis as the result of severe gonorrhœa, the inflammation having extended to the bladder and ureters.

A mild form of catarrhal pyelitis is a common complication of Bright's disease. It sometimes appears during pregnancy, and during the later stages of typhus, as well as during the desquamative period of the acute exanthemata. A hæmorrhagic form of this affection sometimes accompanies scurvy, and the morbus maculosus of *Werlhof*.

Anatomical Appearances.—In croupous or diphtheritic inflammation of the pelvis of the kidney, its mucous membrane is found either to be covered by membranous exudation, or else is converted into diphtheritic eschars, which, upon separation, produce irregular losses of substance.

In recent cases of catarrhal pyelitis, the mucous membrane is reddened by injection, and (especially in the scorbutic form) by ecchymosis. At the same time, it is relaxed, and covered by purulent mucus. When of longer standing the redness disappears, or becomes discolored, the tissue of the mucous membrane is swollen, and, in some cases, is incrusted with urates, or with salts of lime. The pelvis of the kidney usually is dilated, and its walls are thickened. Not unfrequently, the renal parenchyma has suffered atrophy, as in hydronephrosis. In cases of long-continued mechanical irritation, there often is ulceration of the mucous membrane, which may terminate in its perforation, followed by infiltration of urine into the surrounding areolar tissue, with suppurative destruction of the latter. The resulting abscesses may point in various directions, as into the cavity of the peritonæum, or externally, forming long fistulous openings; or, previously forming adhesion, it may burst

into some neighboring organ (such as an intestine). Now and then the liquid contained in the pelvis of the kidney is gradually absorbed. The pelvis shrivels into an indurated tissue, which incapsulates its inspissated contents, the ureter becoming obliterated and converted into a tendinous cord.

Symptoms and Course.—Croupous and diphtheritic pyelitis are scarcely ever recognized during life, as in the majority of cases they are only partial manifestations of those grave and wide-spread disorders which attend the malignant infectious diseases, septicæmia, scarlatina, small-pox, or the typhoid stage of cholera. Sometimes when catarrhal calculous pyelitis becomes aggravated into the croupous or diphtheritic form, shreds and lumps of fibrin are discharged with the urine

Acute catarrhal pyelitis may commence with a rigor or with repeated chills, and be attended by febrile symptoms. The patients complain of pain in the region of the kidney, which radiates along the ureters, toward the testicles, and which is increased upon pressure. When the inflammation has been severe, I have also repeatedly observed vomiting, even in the cases where the disease had spread from the urethra to the pelvis of the kidney. A frequent and sometimes painful inclination to pass water is a constant symptom. According to *Oppolzer*, the secretion of urine increases; this may to some extent be due to hyperæmia of the kidney, in the parts supplied by the vasa afferentia; but if the pyelitis be complicated with interstitial nephritis, the secretion of urine may be diminished in amount. Very often at the outset the urine contains blood, in greater or smaller quantity; and it always contains pus, and in some cases, but not in all, tessclated epithelium, lying row upon row, detached from the pelvis of the kidney. If the disease be of long duration, still more pus appears in the urine. When recently passed, the latter is opaque; after standing a while, a well-defined sediment is deposited, of a yellowish-white color. Owing to the pus serum which it contains, the supernatant liquid shows the characteristic coagulation of albuminous urine upon the application of nitric acid and heat. The microscope shows the sediment to consist of innumerable pus-cells. In pyelitis calculosa, affecting one side only, the impaction of a calculus sometimes causes a temporary but complete arrest of the flow of urine from the pelvis of the kidney. Any urine which is then discharged from the bladder must, therefore, proceed from the healthy kidney. Hence, as long as the obstruction continues, the urine is usually quite limpid, although the symptoms are all aggravated. When the obstruction is removed, the urine again becomes turbid. One patient, whom I have seen, had learned, by experience, that her suffering would be more severe as long as her urine remained clear; and she used to long for the time when pus would begin to pass again.

Owing to persistent suppuration, and to the slow fever which generally accompanies it, chronic pyelitis may lead to marasmus and dropsy, terminating in death. When due to stones in the renal pelvis, the chronic disease is liable to acute exacerbations, particularly after any jolting of the body; or else periodical hæmorrhages occur, which accelerate the exhaustion of the patient. Violent pain in the back, difficulty of motion of the spinal column, pain on drawing up the thighs, aggravation of the fever, and repeated chills, are indicative of threatening perforation of the wall of the renal pelvis. After perforation has actually occurred, and when an abscess has formed in the surrounding tissues, the disease assumes the characteristics of perinephritis. A tumor which fluctuates more or less distinctly is found in the region of the kidney, etc. (see Sec. I., Chap. VII.). When the abscess bursts into the peritoneal sac, the patient speedily perishes, with the symptoms of acute peritonitis. If the pus burrow downward, a cold abscess forms, which makes its appearance in the lower part of the back, beneath Poupart's ligament, or in the perinæum. If the perforation take place into the intestine, masses of pus are discharged with the stools; the tumor which had formed diminishes or disappears entirely, and a more or less permanent recovery takes place.

It is not always easy to decide if a pyuria originate in the bladder or in the pelvis of the kidneys. In a pyelitis without implication of the bladder, the desire to micturate may be very troublesome, and the act of urinating may be accompanied by pain in the urethra. In chronic pyelitis as well as in chronic cystitis, the urine contains quantities of pus-cells, which render it cloudy, and when it stands for some time they sink to the bottom as a whitish-yellow sediment. The idea that the acid reaction of such urine indicates a pyelitis, while alkaline reaction speaks for a cystitis, is not absolutely correct; for in chronic vesical catarrh even when of long standing it is generally acid, and only becomes alkaline in the bladder under certain circumstances, of which we shall speak when treating of catarrh of the bladder. Another criterion also, which I formerly considered valuable in the diagnosis of pyelitis from cystitis—namely, that in inveterate vesical catarrh the purulent sediment was usually mixed with quantities of mucus, while in chronic pyelitis the urine did not contain a trace of mucus—is not always true. The mucous masses evacuated in the urine in some cases of chronic vesical catarrh are the result of mucous transformation induced in the purulent secretion from the vesical mucous membrane by alkaline fermentation of the urine. If the urine does not undergo this decomposition in the bladder, when freshly passed, it will contain no mucous masses; but, if we leave urine with a simple purulent deposit (whether it come from the bladder or the pelvis of

the kidney) in an open vessel till it undergoes alkaline fermentation, the sediment will become tough and mucous. We can only be sure of disease of the pelvis of the kidney, when we find connected groups of spindle-shaped flat epithelium in the urine, especially after violent exercise. But, even in cases where the patient complains of pain in the region of the kidneys, extending thence to the pelvis and testicles, we must not be misled, by any existing dysuria, into the diagnosis of pyelitis.

Treatment.—For acute catarrhal pyelitis we may refer to what has been said already concerning acute interstitial nephritis. At the outset, the application of leeches, cut cups, as well as of cold, over the region of the kidney, is advisable; and, when the pain and strangury are very severe, the exhibition of opium and the subcutaneous injection of a solution of morphine. In order to dilute the urine as much as possible, the patient should drink freely of liquids, avoiding the use of salted or spiced food or beverage. The employment of alkalines, and the alkaline-saline mineral waters of Vichy and Karlsbad is deservedly in repute in the treatment of pyelitis. If the malady assume a tedious form, let the patient use long-continued warm baths, and apply hot poultices to the lumbar region. The acratothermæ are coming more and more into use in the treatment of pyelitis and its kindred complaints. At the baths and springs of Wildbad, I have seen the utmost benefit result from making the patient spend as long a time in the bath as possible, and causing him to drink as much of the pure lukewarm water of the springs as he could take without inconvenience. We do not attempt to decide whether camphor, which enjoys so high a reputation as a remedy for all irritations of the urinary passages, really exerts the beneficial action ascribed to it. Where there is tedious suppuration, astringents should be employed, especially tannin, for reasons already given. In order to diminish the production of pus, we may also try whether the patient will bear the exhibition of the balsams, especially copaiba. For this purpose, *Oppolzer* recommends the patient to drink lime-water and milk (aquæ calcis, milk, āā lb. ½ to lb. ij. daily).

CHAPTER III.

STONY CONCRETIONS IN THE PELVIS OF THE KIDNEY.

[Etiology and Pathology.—Stony concretions may form in any part of the urinary passages, from the uriniferous tubules to the bladder. They may assume the shape of a fine powder (sand or

gravel), or form masses varying in size from that of a mustard-seed to that of the fist. They are classified, according to their composition, into—1. Those composed of pure uric acid, or of uric-acid salts; 2. Those composed of oxalate of lime; 3. Stones of phosphate of magnesia and ammonia and phosphate of lime; 4. Stones of carbonate of lime; 5. Cystine calculi; 6. Xanthine stones. Besides these, there are stones of several components, which are often so formed that around a nucleus alternating strata are superimposed, which vary as the conditions for the excretion of the different materials have varied from time to time. The most common of these stratified stones are composed of a nucleus of uric salts or of oxalate of lime covered by shells of phosphatic matter.

The causes of urinary calculus are many, and in part still unknown. The formation of uric-acid stones is ascribed to the long-continued over-acidity of the urine, due either to the actual increase in the quantity of the acid in the body as a result of imperfect oxygenation, to a sluggish mode of life, to a diet too rich in flesh, or to lack of the supply of oxygen, or else to such an increase of the free acids of the urine that the soluble uric salts which it contains are decomposed, and the uric acid separated. This may proceed from an acid fermentation within the organ, in which the coloring and extractive matter of the urine turn into lactic acid, or else an acidification in the primary passages favors such an excretion. Conditions which produce a concentrated urine, as abstinence from water, persistent diarrhœa, or badly-compensated heart-disease, may also give rise to such excretion.

Oxalate-of-lime stones, generally called mulberry calculi, have likewise various modes of origin. Sometimes, when the urine is alkaline, the acid forms in the urine itself, probably through decomposition of the uric acid. Sometimes the stone forms when the diet has been exclusively vegetable, from excessive use of sorrel, tomatoes, and the like. Again, these calculi may appear under conditions of body not fully understood; but, as we know, the characteristic crystals of oxalate of lime are found in the urine of many patients of sluggish habit, disordered digestion, or derangement of the respiratory or nervous apparatus.

Stones of phosphate of lime or ammonia, or of a mixture of the two, are apt to form wherever a long-continued alkaline condition of the urine produces alkaline precipitates in it. Such decomposition of the urine may proceed from chronic inflammation or other diseases of the urinary tract, due to retention of urine. It may be caused by the use of foul catheters, or by the presence of a foreign body, such as a uric-acid or oxalate-of-lime calculus, which thus may

furnish a nucleus for a phosphatic shell. From the experiments of *Studensky*, who inserted foreign bodies into the bladders of dogs and put lime into their food, it would seem that an excess of lime in the food predisposes toward phosphatic calculi. Even the phosphates which properly exist in the body may by morbid and excessive transformation produce stones in the urine, as sometimes happens in osteomalacia.

Gravel and stones form at all ages of life. In childhood, however, the disposition toward stones—perhaps through uric-acid infarction of the new-born—seems the greater ; and in old age it is also common. That men should suffer more often than women would seem to be because of their different modes of life. How much geological and climatic influences have to do with endemic tendency to stone in certain countries, as England, Holland, and parts of Germany, has not been made clear.

Concretions rarely form in the kidney-substance proper ; but cases like those of uric-acid infarction of the new-born have been seen in the adult, in which uric acid was found after gout, hæmatoidin crystals after hæmorrhage, and bile-pigment after jaundice. Carbonate of lime, too, has been met with. Real stones are also found as very great rarities in the kidney proper, or rather in portions of the dilated uriniferous tubules. The ordinary seat of gravel and of stone is in the calyces or pelvis of the kidney. The smaller stones are roundish ; larger ones mould themselves to the shape of the region which they occupy, so that stones of the calyx acquire a pyramidal form.]

Stony concretions form in the pelvis of the kidney, probably under the same conditions which cause them to form in the bladder. When treating of vesical calculus, we shall discuss the pathogeny and etiology of these calculous formations more fully. Many, and perhaps the majority of stones in the bladder, originate in the pelvis of the kidney, and pass thence through the ureters into the bladder, where they increase in size by precipitation from the urine.

Anatomical Appearances.—In cases of uric-acid infarction, upon section through the pyramid, we see delicate yellowish-red stripes running in the direction of the tubules. Upon microscopic examination, these tubules are found to be filled with a blackish, coarsely-granular material. Upon the addition of an acid, these masses disappear, and are replaced by crystals of uric acid. According to *Virchow*, dilatation of the tubules and the formation of cysts in the kidney of the fœtus may arise from permanent obstruction of the efferent tubules. In the hæmorrhagic-pigmentous infarction we also see red streaks, with a tinge of yellow in them, in

the pyramidal substance; and, under the microscope, the tubules are seen to be filled with granular, or globular, or lumpy masses, and with the well-known hæmatoidin crystals. In calcareous infarction, whitish-yellow stripes are found in the pyramids; and, under the microscope, black masses are visible in the tubules, which dissolve with effervescence upon the addition of acid. The deposits of triple phosphate, which also produce yellowish-white stripes in the pyramids, sometimes coalesce into stones of the size of a hempseed, and may cause suppuration and destruction of the renal parenchyma.

Calculous concretions which form in the pelvis and calyces of the kidney have the same chemical composition as vesical calculi. There is great variety in their size, shape, and number. The smaller ones are no larger than a grain of sand, and are of a rounded form, or else have the shape of a calyx of the organ. The larger ones, which may be of the size of a pigeon's or hen's egg, fill up the whole renal pelvis, often forming a complete cast of its shape, and of that of the calyces.

Symptoms and Course.—Red gritty deposits upon the napkins of newly-born infants, which cease to appear in the course of a few weeks, constitute the sole and not very trustworthy sign of the existence of uric-acid infarction. That of calcareous infarction and the presence of phosphatic deposit in the tubules cannot even be suspected during life.

In many instances stony concretions in the pelvis of the kidney give rise to no symptoms whatever. We often enough see small urinary calculi, which certainly must have formed in the kidney, voided with the urine, the patient never having suffered the slightest inconvenience prior to their discharge. In other instances there is renal hæmorrhage, and in others the signs of pyelitis calculosa. (See Chapter II.) Some persons experience a sense of weight and of pain in the lumbar region whenever the body is jolted. As these symptoms are very ambiguous, we are not warranted in inferring even the probable existence of a renal calculus from them, unless from time to time the pain grow worse, with slight attacks of fever, and unless, after such exacerbations, the urine contain fine granular sediment, or small fibrinous clots incrusted with crystals, and a few blood-corpuscles, or perhaps perfect calculi.

In two of my cases, the diagnosis of renal calculus was strengthened by the fact that, on examining some flocculi which swam in otherwise limpid urine, groups of connected epithelial cells, of the characteristic form of epithelium of the pelvis of the kidney, were recognized; and they showed no signs of fatty metamorphosis, and

had sharp outlines and distinct nuclei, which rendered it probable that they had been detached by some mechanical force.

The passage of a calculus through the ureter is sometimes attended by symptoms to which the term renal colic has been applied. Clots of blood and parasites passing through the ureter may likewise give rise to renal colic ; and it is not impossible, though very improbable, that a mere spasm of the ureter may sometimes have like effect, but such cases are comparatively rare. It is incomprehensible why large stones often pass unnoticed through the ureter, while much smaller ones (which are not always rough or angular) may cause intense anguish. In this affection, the patient (often after a severe jolting) suddenly feels a frightful pain darting from the kidney toward the bladder, and thence to the thigh and testicle. He screams and writhes with agony, and grows cold, prostrate, and demoralized. Though constantly striving to micturate, but little water flows. Vomiting is frequent, so that the seizure is apt to be mistaken for biliary colic or enteralgia. In irritable subjects there may be convulsions. Sometimes temporary remission interrupts the torment of the patient—soon, however, to be followed by renewed and violent exacerbations. In other cases the pain increases steadily, and then ceases as suddenly as it commenced ; and, unless the calculus give rise to fresh trouble by its presence in the bladder, a complete recovery ensues immediately. Such attacks may pass off in a few hours, and rarely last over twenty-four hours ; and, in spite of the formidable aspect of its symptoms, life is hardly ever endangered by renal colic. Sometimes, though not often, the improvement is incomplete ; the pain diminishes, but does not cease entirely, the symptoms of colic becoming complicated with those of pyelitis.

Treatment.—The treatment of uric-acid and calcareous infarctions is, of course, out of the question. In discussing the treatment of vesical calculus, we shall speak of the procedures theoretically proposed for the purpose of dissolving stones, and shall then point out their inefficiency.

In treating renal colic, it is merely necessary that the physician be sure of his diagnosis, in order to obtain the happiest results. He must not content himself by applying leeches, giving restoratives for the small pulse and cool skin, or with other useless half-measures, but should boldly order opium every hour or two. The anæsthetic action of the opium alone is insufficient to account for the relief which usually follows this treatment. It is much more likely that the muscular fibres of the ureter, which have been thrown into a state of contraction by the irritation of the stone,

which they have spasmodically grasped, are relaxed by the narcotism induced by the opium. If the opium given internally be vomited, a subcutaneous injection of morphine may be used. Inhalation of chloroform vapor seems to have an action similar to that of the opium, and is the next best remedy. In like manner, a copious blood-letting may be of service, from the relaxation to which it gives rise; but in most cases this may be dispensed with. The patient, however, who is demoralized by pain and terror, will not be satisfied by the mere hourly administration of a powder and the inhalation of chloroform. He will loudly demand that something more be done for him, and, as a matter of humanity, we should yield to his entreaty. If circumstances permit, let him make use of a warm sitz-bath or complete bath. It has also often been recommended to seat the patient naked upon a stool, and to lay his bare feet upon a cold floor. As a beverage, copious draughts of some mineral water, containing carbonic acid, should be prescribed, in order, if possible, that the urine pressing after the stone may drive it before it into the bladder.

CHAPTER IV.

CARCINOMA AND TUBERCULOSIS OF THE PELVIS OF THE KIDNEYS AND OF THE URETERS.

Carcinoma of the urinary passages is rare. It scarcely ever occurs except where cancer of other organs, especially of the kidney, extends into the walls of the renal pelvis. In rare instances, independent nodules of cancer appear in the pelvis of the kidney and ureters, accompanying carcinoma of the bladder or kidney. Tubercle of the uriniferous apparatus has already been alluded to in treating of tuberculosis of the kidney, and was there stated to be a partial manifestation of a general disposition to tubercle, involving the testicle, prostate, seminal vesicles, and kidney. The diffuse caseous decomposition, which the mucous membrane of the urinary passages presents, cannot always be referred to the formation and softening of discrete tubercular granulations; although sometimes nodules as large as a millet-seed, either in groups or discrete, and (where these have broken down) round or irregular ulcerations, are found upon the mucous membrane. Sometimes the uniform proliferation of cells and their caseous metamorphosis convert the inner wall of the ureter and of the pelvis of the kidney into a yellow pultaceous mass. In other places the degenerated mucous

membrane is fissured and broken down, containing extensive losses of substance. Under such circumstances the urinary passages are always much dilated, and their wall thickened. Tuberculosis of the uriniferous apparatus is easy of diagnosis, when it can be ascertained by palpation that the prostate and testicle also are diseased, and when the urine is alkaline and mingled with pus and *débris*. Extension of tuberculosis from the bladder to the ureters is so common an occurrence that it may always be anticipated in such cases.

SECTION III.

DISEASES OF THE BLADDER.

CHAPTER I.

CATARRH OF THE BLADDER—CYSTITIS CATARRHALIS.

Etiology.—Catarrh of the urinary bladder may arise: 1. From direct irritation of the vesical mucous membrane. Unskilful injections in gonorrhœa, frequent or rude catheterization, foreign bodies, especially calculi, may also produce the affection. The admixture of acrid substances in the urine, or the formation of irritating matter in the bladder, owing to decomposition of the urine, may give rise to a most intense form of catarrhal cystitis. To this class the vesical catarrhs belong, which are induced by the misuse of cantharides and of balsam copaiba, as well as those which arise in cases of spinal disease, typhus, stricture, and enlargement of the prostate, and other conditions causing retention of urine. In the same way, drinking of new beer will occasion a very transient catarrh of the bladder.* 2. In other cases catarrhal cystitis arises from the extension of an inflammatory irritation from other organs to the bladder. First under this heading stands the cystitis which accompanies inflammation of the prostate, and in many cases of gonorrhœa, and which often long outlasts the original disease. 3. Chilling of the skin, especially of the feet and abdomen, not unfrequently results in catarrh of the bladder. 4. Finally, a symptomatic vesical catarrh is one of the concomitants of growths and other grave disorders of the bladder.

Anatomical Appearances.—In acute cases the mucous membrane of the bladder is reddened, swollen, and relaxed. Its surface is covered with mucus, mixed with detached epithelium and young cells in variable quantity.

In chronic catarrh of the bladder this redness of the mucous membrane fades, and the color sometimes becomes a dirty gray. The mucous membrane is swollen and thickened. The submucous and inter-

* Or is this annoying but harmless and transient malady due to acute irritation of the urinary passages by large sharp crystals of oxalate of lime, which may appear after drinking bad beer containing much carbonic acid? I have often urged my students (but hitherto in vain) not to miss an opportunity of examining microscopically the urine passed during such an attack.

muscular connective tissue, and the muscular fibres themselves, are also thickened and hypertrophied. Upon the inner surface of the bladder there lies a gray puriform mucus, or a yellowish purulent secretion. The urine contained in the bladder is often decomposed, is of an acrid ammoniacal odor, and alkaline reaction.

Chronic catarrhal cystitis rarely terminates in recovery. Far more frequently, as the malady advances, the proliferation of cells upon the free surface of the bladder is complicated with a vast cell-growth in the tissues of its mucous, submucous, and muscular coats. In this way catarrhal mucous ulcers and submucous abscesses are formed. Perforation of the vesical wall sometimes takes place. Should this occur after the bladder and its neighboring organs have become consolidated by pericystitis, abscesses may form around the former, which, under favorable circumstances, may open either externally, or into the rectum, or vagina, and, in unfavorable ones, may break into the abdominal cavity. Another, but not a very common, result of vesical catarrh is diffuse suppuration. This generally occurs in cases of retention of urine, and arises from the corrosive action of the putrid contents of the bladder upon its mucous membrane. The mucous membrane is then found to be extremely soft, discolored, and of a brownish-red or blackish hue. It is covered with dirty exudation, or broken down into a ragged, pultaceous substance, infiltrated with fetid pus. The muscular fasciculi are pale and tear easily. The submucous and intermuscular tissue is infiltrated with ichorous matter; within the bladder there is a blackish-brown, chocolate-like liquid, of an intensely ammoniacal odor, consisting of decomposed urine, blood, pus, and shreds of detached mucous membrane. In the worst cases the destruction attacks all the coats of the viscus, whose contents escape into the abdominal cavity.

An enormous thickening of the walls of the bladder, a common result of the hypertrophy of the muscular fasciculi already referred to, may be mentioned as a third termination of chronic vesical catarrh. This condition, however, sometimes arises in cases of long-standing impediment to the passage of the urine, without vesical catarrh. In such cases the wall of the bladder attains a thickness of from several lines to half an inch or more. The muscular fasciculi have grown into rounded bars, and form rib-like projections upon the interior, the appearance of which has been compared with that of the right ventricle of the heart (*vessié à colonne*). According as the capacity of the bladder is increased or diminished by this hypertrophy, it is called excentric or concentric. In the former case the bladder may rise into the abdomen as far up as the navel; in the latter it may be shrunken to the size of a walnut. In some cases of chronic vesical catarrh, es-

pecially when associated with impediment to the exit of urine, the mucous membrane, pressing asunder the muscular fasciculi, is forced in between them, forming *diverticuli*. At first small and round, these latter ultimately expand into large bottle-shaped pouches, of the size of a fist. Their communication with the bladder is at first a narrow chink, which afterward becomes round and sphincter-like. Owing to the incompleteness with which these diverticuli are evacuated, they often become the seat of urinary deposit, and of incapsulated calculi.

Symptoms and Course.—Acute catarrh of the bladder is sometimes accompanied by febrile symptoms. As a rule, however, there is neither elevation of temperature nor acceleration of pulse. In quite recent cases, the patients complain of an undefined pain in the hypogastric region and perinæum, which extends upward toward the kidney, and along the urethra toward the glans penis. In the more severe forms of vesical catarrh, pressure exerted upon the region of the bladder gives rise to pain. The hyperæmic and irritable vesical mucous membrane evinces the utmost intolerance against its contents. The collection of a few drops of urine in the bladder occasions the most urgent desire for its expulsion. The sphincter vesicæ also is in a constant state of spasm, thus causing a vesical tenesmus quite analogous to that of the rectum already described in catarrhal rectitis. The patient scarcely has the urinal out of his hand, micturition is extremely painful, and the few drops of urine which are expelled in short spirts from the urethra produce a feeling as though molten lead were running through the penis. As in all recent catarrhs, at first the quantity of mucus formed is small, so that but a few flocculi are scattered through the urine. Afterward the urine passed becomes turbid, and lets fall a mucous sediment in greater or less profusion. The disease may run its course, and get well in a few days; and that form caused by drinking fresh beer usually passes off within a few hours. In other cases it is more protracted, or passes into the chronic form. Sometimes the spasmodic contraction of the sphincter vesicæ, which occurs in acute vesical catarrh, gives rise to complete retention of urine, and in old persons especially, owing to secondary diseases (œdema and fatty degeneration of the muscles of the bladder), a myopathic palsy of the detrusors arises, which also causes retention. It is this circumstance which renders simple vesical catarrh a dangerous disease to old persons. In chronic catarrh of the bladder, the pain usually abates after a while, but the intolerance of the viscus against its contents and the constant inclination to urinate continue. The quantity of mucus increases considerably. At first a somewhat transparent stratum of mucus sinks to the bottom of the vessel, afterward the urine becomes thick and turbid, and its sediment is more

opaque, and of white or yellow color. If the urine undergoes the so-called alkaline fermentation in the bladder—the secretion of the diseased mucous membrane, full of pus-cells and disintegrating epithelium, suffers a peculiar transformation (already mentioned when treating of pyelitis) forming a gelatinous coherent mass, which, when poured from one vessel to another, holds together, and can be drawn into long threads.

In previous editions of my text-book I have stated that this profuse secretion of mucus often acted as a ferment upon the urine, giving rise to an "alkaline fermentation;" that in this alkaline fermentation the urea was decomposed into carbonate of ammonia, and that new ammoniacal combinations formed, namely, urate of ammonia and phosphate of ammonia and magnesia (triple phosphate). I was compelled to add that the mucus contained in the urine did not always act as a ferment; and that, in many cases of protracted vesical catarrh, I had constantly found the reaction of the urine to be acid. In the course of the last year, from observations made by *Traube*, as well as from investigations and experiments of my own, which have been published by *Teuffel*, in the *Berliner Klinischen Wochenschrift*, I have become convinced that this alkaline fermentation is not produced by the mucus, but is owing to the presence in the urine of organisms of a low grade, which probably usually find their way thither through the introduction into the bladder of dirty catheters. A highly interesting observation, made in my clinic, upon a young girl with palsy of the bladder, affords striking evidence of the correctness of this opinion. In the bladder of this patient, who for weeks had been catheterized with an ill-cleansed instrument, there had developed a most typical alkaline fermentation. Her urine, which was of a pungent ammoniacal odor and alkaline reaction, contained the thorn-apple-like crystals of urate of ammonia, large coffin-lid-shaped crystals of ammonio-magnesia-phosphate, with many vibriones and fungi of a low order; but it contained neither cellular elements nor large quantities of mucus. A careful research into the history of the case showed that the patient never had had any symptoms of vesical catarrh. Chronic vesical catarrh drags on for weeks, months, and even for years. It is a remarkable fact, and one difficult to explain, that it usually is accompanied by loss of appetite and by derangement of digestion. The longer the disease has lasted, so much the less are the chances of a perfect recovery.

Ulceration of the vesical mucous membrane is to be suspected when the sediment of the urine grows more and more purulent, when, from time to time, blood is discharged with the urine, and when a slow fever arises, which consumes the strength of the patient. He then

ultimately dies of vesical-phthisis, especially if it be associated with abscess about the bladder, or tedious suppuration from fistulous passages.

When catarrhal inflammation of the bladder passes over into diffuse suppuration, the patient becomes collapsed, his countenance appears sunken, the pulse is small and thready, and the skin cold. The urine is discolored, of a brownish or blackish hue, contains shreds of mucous membrane, and exhales a fetid odor. Even though the wall of the bladder be not completely destroyed, and though there be no escape of its contents into the abdominal cavity, yet malignant peritonitis soon develops, and the patient dies within a few days, of general prostration.

Thickening of the wall of the bladder, from hypertrophy of its muscular fasciculi, may be detected when the viscus also is dilated, by the appearance above the symphysis pubis of a firm tumor, which sometimes extends as high as the navel, or even higher, and which in females is apt to be mistaken for a distended womb. Generally speaking, patients are unable to empty a bladder thus thickened and distended, even though there be no obstacle to its evacuation in the vesical neck or urethra. It is only the excess of urine (for which, so to speak, there is no room left in the already enormously overfilled bladder) that is passed by the patient, or which runs from him involuntarily if the sphincter be palsied. Thus it may happen that, in the course of twenty-four hours, he may pass a normal amount of water, and still retain from two to six pounds or more in his bladder, which can only be removed by means of the catheter. In concentric hypertrophy, the bladder can be felt through the wall of the vagina or rectum, as a hard tumor, which may give rise to all sorts of blunders. In these cases, the bladder being incapable of distention, there is a constant desire to pass water, which does not give the patient a moment's rest; and, when the sphincter is relaxed, there is an incessant dribbling of urine.

Treatment.—The causal indication, first of all, demands the protection of the vesical mucous membrane from the injuries which have occasioned the disease. This is most difficult of accomplishment where rude catheterization or careless injections into the urethra have provoked catarrh of the bladder; as well as where the exhibition of cantharides and the like, or the long-continued use of fly-blisters, or of irritating ointments, or where persistent retention and decomposition of the urine are the irritants which are acting upon the mucous membrane. When it proceeds from the extension of an inflammation of the urethra or womb, the application of a few leeches upon the perinæum, or portio vaginalis, is advisable. If the apparent cause be cold, we should resort to a diaphoretic course of treatment. It is only when

quite recent and of great intensity that the indications from the disease itself call for local blood-letting, which then is better performed upon the perinæum than above the symphysis. In most cases of acute catarrhal cystitis, hot poultices upon the abdomen, and general warm baths, suffice to relieve the symptoms and to bring about a favorable termination. Besides this, we must take care that the urine enter the bladder in as dilute a condition as possible; but, if we strictly forbid the use of all salt and spices, it will be useless to mix oleaginous or mucilaginous materials in the patient's drink. It is best to let the patient drink the artificial or natural mineral waters—the Seltzer, Wildunger, Fachinger, or Gailnauer waters, or soda-water, or lime-water, mixed with equal parts of milk. The semina lycopodii have a peculiar reputation as a remedy for vesical catarrh (sem. lycopod. ℥ ss—to mel. despumat. ℥ jss. f. elect. s. ʒ j every two hours), as has also camphor, where the complaint arises from the abuse of cantharides (camphor pulv. gr. vj, emulsion of almonds, ℥ vj). The employment of small doses of opium in the form of Dover's powder, given at night before bed-time, or in the form of tinctura thebaica, in divided doses, is not only harmless, but a most efficient remedy against pain and vesical tenesmus. The more the pain abates, and the more copious the admixture of mucus and pus in the urine, so much the more urgently are the astringents indicated. The astringent most commonly employed is a decoction of the folia uva ursæ (℥ ss. to ℥ vj, a tablespoonful every two hours). The continued use of tannin is still more efficacious: I have obtained some most happy results from it in cases which seemed almost desperate. In the later stages of acute vesical catarrh, and still more in the chronic form of the disease, the balsams and resins, which are of such striking benefit in catarrh of the urethra, also do excellent service. To this class belong the oil of turpentine, tar-water, Peruvian balsam, and, above all, balsam copaiba, which may be given in capsules of gelatine. If these remedies fail, we should have recourse to local treatment. I have repeatedly made injections with lukewarm water, the temperature of which I gradually bring down to 65° F. after the manner recommended by *Civiale*, and cannot sufficiently praise their efficacy, especially in treatment of women. Astringent injections, among which solutions of nitrate of silver, sulphate of zinc, and tannic acid, are recommended, should be used with greater caution; so too with injections of emulsion of balsam copaiba (ʒ j to ℥ j), the efficacy of which is greatly extolled by some physicians. No modification of this treatment is demanded where ulceration of the bladder is detected. Suppurative destruction of the vesical mucous membrane is quite insusceptible of treatment. In excentric hypertrophy, the bladder must be emptied by the catheter regularly

every eight or twelve hours, and an elastic bandage should be fitted to the abdomen. In concentric hypertrophy, on the other hand, the patient must be charged to retain his water as long as possible, in order gradually to dilate the bladder. It has also been proposed to introduce an elastic catheter, closed by a cork, into the bladder, by which the urine may be evacuated every two or three hours.

CHAPTER II.

CROUPOUS AND DIPHTHERITIC CYSTITIS.

Croupous and diphtheritic cystitis scarcely ever occur, excepting in cases of severe infectious disease—in septicæmia, typhus, small-pox, and scarlatina—and are accompanied by similar inflammation of other mucous membranes. Far more rarely it arises after the abuse of cantharides, after difficult labor, or in consequence of very intense irritation of the bladder from decomposed urine. In this form of inflammation a coagulating exudation of variable thickness and consistence is formed, a portion of it infiltrating the tissues of the mucous membrane, while another portion lies upon its free surface. The process rarely extends over the entire surface of the bladder. More usually it is confined to detached streaks and spots of a rounded form. After separation of the diptheritic slough, there remain losses of substance in the mucous membrane. The disease can only be recognized when whitish membranous coagula are discharged with the urine, with symptoms of severe tenesmus. In the croupous cystitis which sometimes follows the abuse of cantharides, or difficult forceps deliveries, we occasionally see large tenacious false membranes discharged with the urine. The treatment of croupous and diphtheritic cystitis should be similar to what we have already advised in cases of violent and acute catarrhal cystitis.

CHAPTER III.

PERICYSTITIS.

Besides the inflammation induced by perforation of the bladder, and by abscesses and suppuration of its walls, another and independent inflammation, which we call pericystitis, sometimes involves the connective tissue which surrounds this organ, and connects it with the adjacent parts. It is of far less frequent occurrence than the inflammation which takes place about the rectum, and is hardly ever observed excepting as an accompaniment of infectious disease, typhus, the acute exanthemata and septicæmia. As a still greater rarity, it arises idio-

pathically without known cause, in persons previously in good health. This inflammation shows great tendency to pass into suppuration, and to destroy the parts involved; it is very apt to spread into the tissues which attach the bladder to the other pelvic viscera, and to the sides of the pelvis. The pus may ultimately penetrate into the bladder, rectum, vagina, or externally into the perinæum. There is also a form of chronic pericystitis, which not unfrequently accompanies chronic vesical catarrh, and vesical ulceration, and which creates induration of the surrounding connective tissue, and causes firm adhesion of the bladder and surrounding parts. Sometimes this also results in the formation of an abscess.

The affection is generally difficult of recognition, as the resulting painful tenesmus of the bladder, the dull, continuous pain in the pelvis, the repeated chills, and the complete retention of urine, which occur when the urethra or the ureters are obstructed by an abscess, furnish insufficient data for a diagnosis. A diagnosis can only be in some degree certain when an abscess projects above the pubis from the anterior surface of the bladder, presenting a spherical prominence, which does not disappear when the bladder is emptied; or else when we can feel a tumor in the perinæum, or through the rectum or vagina. The treatment of pericystitis belongs to the province of surgery.

CHAPTER IV.

TUBERCULOSIS AND CARCINOMA OF THE BLADDER.

Tuberculosis of the bladder usually appears as a complication of tuberculosis of the ureters, renal pelvis, and kidneys. Discrete and conglomerate tubercles also form in the bladder sometimes, which, upon breaking down, produce rounded or irregular excavated ulcers. Sometimes (but more rarely than in the ureters and pelvis of the kidney) there is a diffuse caseous degeneration of the mucous membrane, which occasions wide-spread destruction.

The symptoms of tuberculosis of the bladder are very similar to those of chronic vesical catarrh and vesical ulceration. The copious admixture of mucus and pus in the urine, which is often in a state of ammoniacal decomposition, the tormenting desire to urinate, the frequent hæmorrhages from the bladder, which are the signs of excentric and concentric vesical hypertrophy, also attend vesical tuberculosis. Our only positive data for a differential diagnosis are the coexistence of degeneration of the prostate or testicle, and the appearance of elastic fibres, or of larger bits of tissue in the urine, from which we may infer the destructive nature of the process which is going on. In a female

patient of mine, suffering from tuberculosis of the bladder, urinary passages, and kidneys, I found an irregular eroded ulcer, with peculiar edges, in the vulva, surrounding the orifice of the urethra.

The treatment of tuberculosis of the bladder is identical with that of chronic vesical catarrh, but is generally quite unavailing.

Carcinoma of the bladder is not common. Sometimes it is primary, sometimes it occurs secondarily to similar disease of the uterus or rectum. It assumes the form of scirrhus, more often the medullary form, but the villous cancer is more frequent than either. The former two sometimes produce diffuse degeneration of the wall of the bladder, which, upon breaking down, forms communicating openings with the vagina, uterus, or rectum. Sometimes they take the shape of circumscribed and even pedunculated growths. The villous cancer forms soft tumors, consisting of thin, delicate villous excrescences, which float in water, and which often become detached during life, giving rise to hæmorrhage.

The most prominent symptoms of cancer of the bladder are, likewise, those of chronic vesical catarrh. Hæmorrhage is still more common than in tuberculosis. Diagnosis must depend upon the continuance and constant aggravation of the symptoms, the early appearance of a bad cachectic condition, in the discovery of cancer of other and especially of neighboring regions, and, above all, upon microscopic examination of the detached particles of the growth discharged with the urine. Treatment is in vain, and should be limited to a repression of the hæmorrhage, the relief of any retention of urine which may arise, and the combating of intercurrent symptoms.

CHAPTER V.

HÆMORRHAGE FROM THE BLADDER—HÆMATURIA VESICALIS.

Hæmorrhage from the vessels of the bladder is often of traumatic origin. Sharp-edged stones, or foreign bodies, which have entered the viscus, are the usual causes of the bleeding. In hysterical women, we must be prepared for the most extraordinary devices. Not at all unfrequently, they introduce foreign bodies into their genitals, or into their urethra, which may be the cause of the hæmorrhage. In other cases, ulcers of the bladder cause erosion of its vessels, and consequent bleeding. Neoplastic formations also, tubercle, carcinoma, but especially the villous cancer, give rise to losses of blood. Very rarely, such hæmorrhages depend upon the misuse of cantharides (hæmaturia toxica), or upon a hæmorrhagic diathesis. Finally, an excessive dilatation of varicose veins of the part may result in their rupture, with

an effusion of blood. Hæmaturia from this cause, however, is very rare, although the laity are much inclined to ascribe all hæmaturia to "hæmorrhoids of the bladder." The conditions producing impediment to the circulation and dilatation of the vessels are far less favorable in the bladder than in the rectum; and, as a rule, this last and rarest of the causes of vesical hæmorrhoids is not to be thought of, unless, after a scrupulous review of all the symptoms, all other forms of the disease can be excluded from the diagnosis.

In hæmorrhage into the bladder, the blood and urine are less intimately mixed than when the bleeding comes from the ureters, renal pelvis, or kidneys. The clots which form are also of larger size. Nevertheless, both these signs may prove fallacious, and hence diagnosis of the region whence the bleeding proceeds is often a matter of great difficulty. The attendant symptoms furnish the most trustworthy aids to diagnosis of the source of the hæmorrhage. Vesical hæmorrhage is almost always accompanied by evidence of change of structure in the bladder. During the intervals between the bleedings the urine contains mucus and pus, and there is dysuria, etc.

Our main task in the treatment of vesical hæmaturia should be to combat the original disease. In severe cases, cold should be applied to the region of the bladder, and large doses of tannin should be given. Where the bleeding threatens to exhaust the patient, we must have recourse to injections of cold water, with a solution of alum, sulphate of zinc, or nitrate of silver. In vesical hæmorrhoids as well as other forms of vesical hæmorrhage, cold injections have the best effect.

CHAPTER VI.

STONY CONCRETIONS IN THE BLADDER.

Etiology.—The manner in which stones form in the kidney and bladder is obscure. None of the current explanations of the process have shown themselves proof against the various objections made to them. Especially is this the case with the theory that these concretions are the result of a peculiar derangement of assimilation, of a diathesis, wherein uric acid, oxalic acid, the phosphates, etc., form so profusely in the system, and are eliminated from the blood through the kidneys in such quantities, that they are precipitated in the urinary passages.

The explanation of *Scherer* is very plausible. He believes that the substances deposited in the urinary passages, and which sometimes form stones, are not eliminated from the kidneys in their present shape, and only form through the decomposition which the urine undergoes

during its abode in the urinary passages. This decomposition is similar to that which takes place in urine when allowed to stand exposed to the air. There is at first an acid fermentation. The coloring and extractive matter of the urine become converted into lactic acid, so that the urea is liberated from its soluble combinations, and is precipitated. The acid fermentation is followed by an alkaline one. The urea is converted into carbonate of ammonia, and, by a combination of the ammonia with the phosphate of magnesia, an ammonio-magnesian phosphate is formed—the so-called triple phosphate. The ferment which excites this decomposition in the urine, while yet in the bladder, is the mucus resulting from vesical catarrh; but, according to *Scherer*, besides this, the mucus plays another and most important *rôle* in the formation of calculi, by forming a cement to hold the sediment together, since coagula of mucus form the nuclei of most stones, to which the deposits afterward adhere by accretion. According to the theory of *Scherer*, the formation of stones, which consist of a nucleus of uric acid enclosed in layers of phosphates, is as follows: As long as the acid fermentation of the urine continues, uric acid is precipitated; when, however, the catarrh has continued for some time, and, perhaps, has become aggravated by the presence of a calculus in the bladder, alkaline fermentation sets in, and the phosphates are thrown down.

According to a very brilliant hypothesis of *Meckel*, the formation of precipitates is not requisite for the production of a urinary calculus. He claims that almost all stones consist originally of oxalate of lime, and are formed as follows: The mucous membrane of the urinary passages becomes the seat of a specific catarrh, called by *Meckel* the "stone-forming catarrh" (stein-bildenden catarrh). In this catarrh a tough adhesive mucus is secreted, which has a tendency to acid fermentation, and in which oxalate of lime appears when such fermentation occurs. At first this oxalate-of-lime mucus is of a gelatinous consistence. Gradually, however, it takes up more and more oxalate of lime from the decomposed urine, and thus, growing more and more firm, finally becomes stony. As long as the urine remains decidedly acid, the stone enlarges, from the accretion and petrifaction upon it of fresh layers of oxalate-of-lime mucus. If the urine afterward become alkaline, the stone no longer grows from "apposition," but from "intussusception," combined with a "metamorphimus," that is to say, the oxalate of lime is at first displaced by uric acid, and urate of ammonia, and afterward by the phosphates. In this manner an oxalic calculus becomes converted into a phosphatic calculus.

To attempt to point out all the flaws in each of these theories, and the various objections which might be advanced against them, would take too long. Among other matters, the fact still remains unex-

plained, that hereditary tendency should play so decided a part in the formation of stone in the bladder. All the different generations of one family have been observed to suffer from the same sort of stone. Males are attacked by stone more frequently than females. The age of childhood is by no means exempt; indeed, children are rather liable to the formation of calculi and gravel in their urinary passages. In some countries, as in England, the malady is much more common than in others. Drinking-water which contains lime seems to have some influence in the production of stone, and the use of fermenting beverages containing carbonic acid is quite decided, at least as to its effect in producing oxalate-of-lime calculi. Any irritation which occasions catarrh of the urinary passages may give rise to stone in the bladder; but it is quite enigmatical why many catarrhs last so long without forming concrements, while others give rise to their formation very soon.

Anatomical Appearances.—Urinary calculi vary in size, shape, and chemical composition. The smaller, which are usually extremely numerous, we call "gravel." Their form and color depend mainly upon their composition. They may be classified as follows:

1. Stones consisting chiefly of uric acid and its salts. They are round or oval; usually of a reddish-brown color, very hard and heavy, and with a smooth or nodular surface.

2. Stones of oxalate of lime. They usually present a nodulated, gland-like surface, whence their name, "mulberry calculus." They are very hard, and of a dark-brown or blackish color (through the admixture of transformed hæmatine). Yet some oxalate-of-lime stones are small and pale, and in form bear a strong resemblance to hemp-seeds.

3. Stones consisting of ammonio-magnesia phosphates. They are of a whiter or grayer color; have a round or oval shape; are light in weight, and are of friable, chalky consistence.

4. Calculi of cystine. These are rare. They are usually of a yellowish-white color. Their surface is smooth, more rarely nodular.

5. Stones formed of xanthin, which are more rare even than the cystine calculi. They are very hard, of a yellowish-red color, and usually have a smooth surface.

6. Stones, consisting of various layers and strata of diverse composition, are very common. Sometimes, the nucleus is of uric acid and the outer layers of oxalate of lime; more rarely, the contrary arrangement obtains. The most common form of stratified calculus is that in which a nucleus of uric acid or of oxalate of lime is enclosed in a hull of phosphates, or in which there are several alternate layers of these substances.

Stones often lie free in the bladder, changing their position as the

attitude of the body is varied. Quite frequently, too, they are lodged and embedded in pouches and diverticuli in its wall. The mucous membrane is either in a state of catarrh or of catarrhal ulceration. The muscular tunic is usually hypertrophied; yet, if the calculus be smooth and light, all of these appearances may be absent.

Symptoms and Course.—Sometimes, but not very often, patients with "stone" have a distinct perception that there is a foreign body in their bladder which changes its place as the posture of the patient is altered. A more important and constant symptom is pain about the bladder, which is aggravated by walking, driving, or riding, and which is relieved by lying upon the back. The pain darts along the penis to the glans, and causes the patient, especially if he be a child, to pull upon the prepuce, so that in children an œdematous thickening or abnormal length of the prepuce is to be regarded as a characteristic, or, at least, as a suspicious sign of vesical calculus. During micturition, the stream of urine is often suddenly interrupted, owing to obstruction of the neck of the bladder by the stone. If the patient change his attitude, his ability to pass water is frequently restored. Even though the beginning of the act of micturition be easy and painless, yet, toward its close, there is usually the most acute suffering. This is combined with aching in the testicles, thighs, and lumbar region, with spasmodic contraction of the anus, and even with general reflex phenomena. The whole of these symptoms, however, together with occasional hæmaturia, are insufficient to establish a diagnosis; and we should make it a rule never to pronounce a positive opinion until the presence of a stone has been reduced to a certainty by means of the sound.

Treatment.—The possibility of dissolving large vesical calculi, by means of internal medication, must be pronounced as hitherto unproved, although theoretically such possibility cannot well be denied. Means have been proposed of rendering the urine alkaline, or else of increasing its acid reaction, according to the chemical constitution of the stone. The former task, as is well known, is far easier of accomplishment than the latter, as the carbonates and vegetable salts supplied to the system are discharged with the urine as alkaline carbonates, while it is quite difficult to augment the quantity of acid in the urine. Theoretically, the benzoic acid, which is excreted in the form of hippuric acid, is adapted for the solution of a phosphatic calculus. But the continued exhibition of this remedy in large doses is forbidden, owing to its pernicious action upon the digestive organs. The administration of tartaric and citric acid might also have a good effect in the solution of phosphatic calculi; but these harmless articles have never come into use. Indeed, as soon as a stone in the urinary passages has been

diagnosticated, it is usual to administer alkaline carbonates, quite regardless of the chemical nature of the stone, and to send well-to-do patients to Vichy or Karlsbad, those springs having a world-wide reputation as remedies against lithiasis. Perhaps the benefit derived from this mode of treatment depends upon the effect of the remedies upon the vesical catarrh, which is one of the main causes of the affection. According to *Meckel's* theory, the exhibition of the alkaline carbonates and salts of the vegetable acids is indicated, because the urine is thereby rendered alkaline, and the metamorphosis of a stone, consisting of oxalate of lime, or of uric acid, into a softer and more friable phosphatic calculus, is promoted. At all events, we strongly advise that the old method of treatment be adhered to, and that no new "cures" be adopted, based merely upon *a priori* reasoning. Besides the alkaline carbonates, and the basic phosphate of soda, of which about two drachms may be taken daily, the carbonate of lithia has of late obtained a great reputation as an antilithic, and is warmly recommended by many. I should attach little weight to such recommendations, were they based solely upon the theoretical ground that the carbonate of lithia has a solvent power of uric acid six times as great as that of bicarbonate of soda. But, as we have some reports, upon good authority, of good clinical results obtained by its use, I admit the propriety of making further experiment with this drug (lithiæ carb. gr. j—v t. d.). The Wildunger, Vichy, or Karlsbad waters may also be used as a beverage.

We have already discussed the mode of treating the catarrh and hæmorrhage of the bladder to which calculus gives rise. The operation for stone in the bladder is a matter for the surgeon.

NEUROSIS OF THE BLADDER.

We are still in need of considerable physiological light as to the normal innervation of the bladder, and to the process of micturition. It is difficult satisfactorily to account for the well-known facts that a healthy man is often unable to pass water when required to do so in the presence of a stranger who watches him, and that the first attempts of a novice to make water while on horseback or in a wagon are often attended with difficulty, while these things are easy enough to one who is accustomed to them. Nevertheless, our comprehension of the nervous disorders of the bladder will be facilitated if we classify them into motor and sensory neuroses, and subdivide the latter class into hyperæsthesia and anæsthesia, the former into hypercinesis and acinesis.

CHAPTER VII.

HYPERÆSTHESIA OF THE BLADDER.

Hyperæsthesia of the bladder is most commonly seen in individuals addicted to sexual excess, especially to onanism—a very moderate degree of fulness of the bladder causing in them a most intense desire to urinate—"*castus raro mingit.*" If they have not opportunity to indulge their inclination to pass water, they suffer pain over the bladder and along the penis. The power of retaining the urine is not generally impaired in such cases, the hyperæsthesia being pure and uncombined with motor disturbance. Sometimes, however, this irritability of the bladder is accompanied by a diminution of power in the detrusors; and it is not without reason that a strong stream of urine passes for a sign of chastity among the laity, and a languid dribbling one for a token of the reverse. Extreme hyperæsthesia of the bladder sometimes follows gonorrhœa. Patients are sometimes met with who are quite unfitted for their previous occupation, being unable to hold their water for over a quarter of an hour at a time, and are thereby reduced almost to desperation. This form of hyperæsthesia, perhaps, is always accompanied by a slight catarrh. The intolerance of the bladder against its contents is the most prominent symptom of this form of catarrh; and I have never noticed any excessive production of mucus.

For the milder form of hyperæsthesia of the bladder, such as affects dissipated persons and onanists, I recommend cold river and sea baths, cold sitz-baths, and cold douches. The more severe forms, which remain after cure of a gonorrhœa—the cold-water-cure, injections into the bladder, and other active procedures, proving ineffective—I have in some instances seen disappear rapidly and without trace under large doses of balsam copaiba.

There are no well-authenticated observations of neuralgia of the bladder, that is, of painful excitement of the sensory nerves, which cannot be traced to irritation of their peripheral extremities.

CHAPTER VIII.

ANÆSTHESIA OF THE BLADDER.

Some persons can suffer the accumulation of a very large amount of urine in their bladder without feeling an urgent inclination to evacuate it. This condition, however, is hardly to be regarded as a morbid one. On the other hand, it seems to me admissible to ascribe the

enuresis nocturna (nocturnal wetting the bed) to an imperfect anæsthesia and diminished irritability of the vesical sensory nerves. This extremely distressing affection, for which the patient not unfrequently is subjected to the most abominable treatment, and which often destroys the happiness of families whose older children are afflicted by it, is usually classed with palsy of the bladder. Many cases of enuresis nocturna as I have seen, however, I have never been able to perceive that the sphincter vesica failed in its duty during the day, or that the children had any dribbling of urine, nor when the inclination to urinate came upon them were they ever in great haste to reach the urinal. Indeed, there are only two possible ways of accounting for enuresis nocturna. Either the sensation to which a full bladder gives rise is too feeble to awaken the child from his normal sleep, or else the sensation is of its normal intensity, but the sleep is unusually profound. In the latter case it would seem as though nocturnal incontinence of urine occurred somewhat as do those well-known cases in which children sometimes fall out of bed in their sleep without awakening. In the cases which I have seen, especially in adult patients, I have not been able to discover such unnaturally profound sleep. Fortunately, it is rare for enuresis nocturna (which usually affects children, and often lasts up to the time of puberty) to continue after the twentieth year. Knowledge of this fact is of importance, the best means of comforting the patient and his relatives in their distress being the communication of this intelligence.

It is customary to deprive children, suffering from this affection, of all drink or liquid food during the evening hours, and to waken them during the night once or twice to make them pass water. There is no objection to be made to these measures, excepting that they scarcely ever do the least good. I must protest most earnestly, however, against the cruelty of whipping or otherwise punishing a patient with nocturnal incontinence, unless the wettings of the bed manifestly be the result of laziness. In families, but still more in boarding-schools, orphan asylums, poor-houses, and prisons, punishment for this offence is inflicted all the year long, generally without effect; and if we only take the trouble to listen to the shocking stories of "piss-a-beds" of the lower classes, it will be clear to any one that the inefficacy of the punishment was not due to its want of severity. Without attempting any explanation of the fact, I will merely call to mind that the dread of going to sleep often has a soporific effect, the desire to do so the opposite, and that, if one wishes to awake at a certain hour, the fear of oversleeping is injurious, while the confidence that one will awaken in time is beneficial. In the same way, we find that children with nocturnal incontinence, who have been severely chastised the day before,

and who go to sleep in dread of further punishment, still awake in the morning with their bed wet through. On the other hand, if we can excite confidence in the use of some entirely inert medicine, for which, however we promise great things, so that the patient can go to bed hopefully, and without fear, he is often awakened in the night by the inclination to micturate, and remains for some time, or even permanently, cured of his affection. From time to time secret remedies for nocturnal incontinence, with testimonials as to their efficacy, are advertised in the newspapers. That such testimonials are not all intentionally false, is certain; but it is equally certain that the success of the remedies is due more to the psychical impression made by the confident commendation of the article, than upon the medicinal effect of the article itself. We must never tire of again and again exciting the hopes of the patient, and of writing harmless prescriptions for him, of the value of which he must be assured. Even in little children, and still more frequently in grown persons, I have seen at first a temporary and afterward a permanent effect produced in this way, which amazed both the patient and his friends. I have usually ordered small doses of carbonate of potash, and recently, according to a suggestion of *Trousseau's*, one-fifth to one-third of a grain of pulv. herb. belladonna, with equal parts of the extract. Other remedies, such as strychnine, cantharides, large doses of syrup. ferri iodidi, and injections of irritating matter into the bladder, are objectionable. It is of importance, however, to pay attention to any indications which may arise from the special condition of the patient.[1]

CHAPTER IX.

HYPERCINESIS OF THE BLADDER—SPASM OF THE BLADDER—CYSTOSPASMUS.

Violent contraction of the muscles of the bladder frequently arises in consequence of the irritation which foreign bodies, especially stones, create within it. Such spasm is usually accompanied by organic disease of the viscus. However, since abnormal irritability of the motor nerves of an organ arising as a reflex symptom, in consequence of excitement of the sensory nerves of that organ from structural disease, is not usually counted among the neuroses, the term spasm of the bladder is not to be applied to such symptomatic contractions of the vesical muscles, but should be reserved to designate the abnormal condition of the motor nerves, which exists independently of any structural alteration in the wall of the bladder.

Romberg divides the causes of spasm of the bladder into cerebral, spinal, and reflex. With regard to the first, I would call to mind the fact that the irritability of the sympathetic nerve, though independent of the influence of the will, is by no means entirely independent of the irritability of the fibres and ganglia of the brain. Mental emotions have a distinct action upon the sympathetic system; and just as the muscles of the skin contract under the influence of terror, producing a "goose-skin," so we often, under like circumstances, see violent contraction of the detrusor urinæ muscles, with intense inclination to pass water. So, too, in inflammation of the spinal marrow and in structural disease of the brain, spasm of the detrusors occurs, according to *Romberg*—a fact which is not easy to explain, as the innervation of the detrusor is derived from the sympathetic; although, indeed, we see an analogous condition in the occurrence of vesical spasm from mental emotion. Vesical spasm is generally of reflex origin. In an irritable subject the impression upon the sensory nerves of the urethra, produced by the introduction of a catheter, will provoke spasm of the sphincter vesica. In other cases, irritation of the rectum and, very frequently, irritation from the womb give rise to spasmodic contraction of the bladder. Finally, there are cases of vesical spasm which are merely local manifestations of a general neurosis, and must be ascribed to a morbidly exalted irritability of the entire nervous system, which we usually call hysteria.

The symptoms of spasm of the bladder vary according as the affection involves the detrusor urinæ or sphincter vesicæ muscles. In the former case, a very slight degree of fulness excites intense desire to urinate. The patient can scarcely prevent a constant escape of water by closure of the sphincter; or else he is quite unable to do so, so that the urine is constantly flowing away—a condition generally termed *enuresis spastica.* On the other hand, when the sphincter muscle is in a state of spastic contraction, the patient can only pass water drop by drop, or in a very fine stream, and with great effort (*dysuria spastica*). Sometimes the closure of the sphincter is absolute, and there is complete retention of urine (*ischuria spastica*). Finally, the antagonistic muscles, both detrusor and sphincter, may be attacked simultaneously. A condition of great distress then ensues, in which, in spite of violent desire to void the urine, there is inability, more or less complete, to satisfy such desire. In these cases the spasmodic affection sometimes extends to neighboring organs, giving rise to spasm of the rectum, to tremors of the entire body, and occasionally to general convulsions. One characteristic of spasm of the bladder is the alternation of violent paroxysms and intervals of exemption. The former often last but a minute or two, although sometimes they last half an hour or more.

They recur at longer or shorter intervals, and usually cease as suddenly as they have begun.

Diagnosis of spasm of the bladder in its stricter sense demands caution, as its occurrence is somewhat rare, and as it is often extremely difficult to distinguish it from other maladies of that viscus. It is only when the latter can be excluded from diagnosis, after careful examination of the urine, and scrupulous attention to the attendant symptoms of the case, and after we have assured ourselves by repeated sounding that there is no foreign body lodged in the bladder, that we are warranted in supposing the existence of a pure vesical hypercinesis.

Treatment of spasm of the bladder, first of all, demands removal of the exciting cause. In some instances, the cure of a fissure of the anus, or of a chronic hyperæmia or inflammation of the womb, constitutes the chief, or, indeed, only remedy, for vesical spasm. In other cases, it will subside if we can succeed in altering the nutritive condition of the patient, by a thorough modification of external relations, and thus quiet the morbid irritability of the nervous system. For the paroxysms themselves we should have recourse to warm baths, clysters of camomile or valerian tea, with the addition of a narcotic; and, above all, to the internal exhibition of opium. In addition to this, *Pitha* warmly recommends the cautious and gentle introduction of a wax bougie into the bladder.

CHAPTER X.

ACINESIS OF THE BLADDER—VESICAL PALSY—CYSTOPLEGIA.

Palsy of the bladder may involve the sphincter muscle, the detrusor urinæ, or both of these muscles simultaneously. Contraction of the detrusor is not dependent upon volition, but arises reflexly from the stimulus of the urine collected in the bladder. Contraction of the sphincter, on the other hand, is subject to the will. Up to a certain point of distention, the tone of the sphincter suffices to counteract the action of the detrusor, which presses upon the contents of the bladder and strives to open its outlet. If this point be exceeded, the tone is overcome, and the sphincter requires the action of volition to keep it closed. From these physiological facts, the causes of vesical palsy become somewhat more comprehensible. In the first place, it is quite plain, both in structural disease of the brain and in grave febrile constitutional disease, in which the function of the brain is overthrown, why palsy of the sphincter should be a very common symptom, and why palsy of the detrusor should be somewhat more rare. In apoplexy and in typhus, the number of patients who wet their bed is greater

than that in which the use of the catheter is required. If, however, the paralysis finally extend from the cerebro-spinal system to the sympathetic; if the power of involuntary movement be also impaired; if the patient can no longer swallow; if the abdomen become tympanitic from palsy of the intestinal muscles; the detrusor urinæ also takes part in the paralysis, and the distended bladder rises above the symphysis. There is another very common circumstance, however, which also contributes to the occurrence of incontinence of urine in cases of disease of this kind, namely (as happens in a variety of conditions of other peripheral organs), the overfilling and overflowing of the bladder (*Kohlrausch*) make no impression on the consciousness, and hence no impulse is derived from the will to contract the sphincter and to close the bladder. At the same time, however, it must be admitted that, in some cases of apoplexy and of severe typhus, palsy of the detrusor sets in early, and before there is any general palsy, and without our being able to account for this phenomenon. In disease of the spinal marrow, likewise, palsy of the sphincter is of far more common occurrence than palsy of the detrusor. Most paraplegic patients have to wear a urinal, in order not to wet themselves, because the conducting power of their spinal marrow is interrupted. The use of the catheter, on the other hand, is but seldom necessary, on account of palsy of the detrusor. However, the fact that such necessity does sometimes occur, perhaps is owing to the locality of the lesion. It seems to be the ganglia which transmit the impression from the sensory nerves of the bladder to the motor nerves. It is possible that, if that portion of the spinal cord be destroyed at which this transmission takes place, the detrusor will be palsied; on the other hand, that, if the lesion of the spinal marrow be above this point, transmission of impressions from the brain to the sphincter will be interrupted, while that from the sensory nerves of the bladder to the motor fibres of the detrusor continues unimpaired. The result of comparison of a large number of cases of this kind, which I have examined within the last few years, fully supports this hypothesis.

Besides this form of vesical palsy, the cause of which is *central*, there are others in which the peripheral expansions of the nerves have suffered lesions, through which they are deprived of their function; although, indeed, it is generally out of our power to furnish anatomical proof of such structural disorder.

Finally, there is a myopathic palsy of the bladder arising from disease in the minute texture of the muscular fibres of the organ, and in the nerve-tips which terminate in them. The most common cause for this form of vesical palsy is the severe stretching of the vesical muscles, and their participation in the affections of the mucous membrane.

A distention of the bladder, which perhaps has occurred but once, and to which a mechanical obstacle to the discharge of the urine, or a false shame in some over-modest person, has given rise, may result in permanent vesical paralysis. It may also proceed from a vesical catarrh, especially in aged persons, from implication of the vesical muscles in the disease.

The symptoms of vesical palsy vary according to the muscles affected. When the palsy is complete and is confined to the sphincter, the urine flows away involuntarily as soon as the bladder has become full enough to overcome the tone of the sphincter. When the paralysis is incomplete, the patient is able to resist the pressure of a somewhat fuller bladder, but he must hasten to find some befitting place to pass water, as his sphincter will fail of its office if he wait too long and the pressure increase. This incomplete palsy of the sphincter is of very common occurrence in conjunction with partial paralysis of the lower extremities, and is attributable to an incomplete interruption of conducting power of the spinal marrow.

In palsy of the detrusor, the tone of the sphincter is not overcome by such a degree of tension of the bladder as, under normal conditions, would excite contraction of the detrusors sufficient to overcome it. Unless artificially emptied, the bladder is distended excessively, and it is only after the distention has reached an extreme point, or by the action of the abdominal muscles (where they are not paralyzed also), that a portion of the contents of the bladder is expelled. If the palsy be pure and uncomplicated, the patient can arrest or postpone the outflow of urine. When incomplete, the bladder does not become so full before evacuating a portion of its contents as it does in complete palsy. The patient, however, endeavors to assist the action of the detrusor, by bringing his abdominal muscles into play, so that he often breaks wind while passing water. In spite of all his efforts, he cannot eject a strong arched stream, but the urine dribbles vertically down between his feet. This form of incomplete palsy of the detrusor is chiefly seen in marasmic subjects, and individuals weakened by sexual excess.

Finally, in patients in whom both sphincter and detrusor are palsied, the bladder is in a state of permanent over-distention, the tone of the sphincter yielding later than it normally should do. Every addition made to the contents of the bladder induces a corresponding outflow from it, and the patient is unable to hinder or to arrest this flow. The patient usually, indeed, is quite unaware that his bladder is full, and only seeks medical aid on account of the continual enuresis, and generally is greatly surprised when we evacuate the urine from the bladder, which often contains an enormous quantity of it.

In treatment of palsy of the bladder, the causal indication can rarely be met. Especially is this the case in the forms of it arising from disease of the brain or spinal marrow. When it is due to immoderate distention of the organ, it is of importance to use the catheter assiduously, partly in order to prevent further distention, which might aggravate the palsy, partly to excite more vigorous contraction of the detrusor, by the irritation which the catheter induces. In cases of incomplete palsy, instead of passing a catheter, *Pitha* recommends the introduction of a solid wax bougie as far as the neck of the bladder, claiming that "the catheter" relieves the muscles of all exercise, and thus encourages "their indolence." The indications from the disease call for the use of cold baths, douches, and clysters. If these fail, and if the source of the palsy be peripheral, we should proceed with due precaution to the injection of water into the bladder, which at first should be lukewarm, and afterward should gradually be reduced in temperature. If this be of no avail, the application of electricity, as recommended by *Duchenne*, will also prove ineffectual. We shall hardly find a well-authenticated case of cure of vesical palsy by the use of strychnine, which has also been recommended.

ADDITION TO THE REVISED EDITION OF 1880.

SECTION III.—DISEASES OF THE BLADDER.

1.—P. 82.

I have now and then earned the thanks of an adult by recommending to him a contrivance which has been advertised in the newspapers. A bit of leather or caoutchouc, of the size of a bean, is to be pierced through its middle by a fine opening, through which the loop of an elastic band is to be drawn, so as to slide stiffly. Upon going to bed, the prepuce, having been stretched over the glans, is to be inserted into this loop, which must then be tightened just enough to close its orifice, but not closely enough to cause discomfort, and so that upon urinating the prepuce will be put on the stretch by the water which is held back. The patient will be awakened by the distention of the prepuce if he passes water during sleep, and may then take off his apparatus, confident that for that night his bed will not be soiled. One young man tells me that only since obtaining this contrivance has he ever dared to sleep away from home.

SECTION IV.

DISEASES OF THE URETHRA.

According to the plan of our book, we omit without notice all affections of the urethra pertaining to the department of surgery, and which are treated of in detail in surgical works, and shall merely describe the inflammation of the urethra.

CHAPTER I.

VIRULENT CATARRH OF THE MALE URETHRA—CLAP—GONORRHŒA.

Etiology.—The urethral mucous membrane does not undergo any specific change in gonorrhœa. The process which takes place in it is identical with that which arises in any other mucous membrane, under the action of other irritants, and bears the name of catarrh or blennorrhœa. Nevertheless, gonorrhœa is a specific disease. Its course distinguishes it plainly from all other catarrhs which affect the urethral mucous membrane, or that of other regions. The difference is all the more especial in an etiological point of view, for a gonorrhœa never arises otherwise than by contagion, in spite of the persistent denial of some authorities, and the lying assertions of shame-faced patients.

Of the nature of the virus of gonorrhœa we know as little as we do of that of small-pox or other infectious matter; but we do know that the matter exerts a specific influence upon the system—that it always produces a clap, never a chancre or syphilitic ulcer. True, opinions are still divided as to whether this disease is followed by secondary disorders, and by a general implication of the system; but even authors who still believe in metastatic gonorrhœa and gonorrhœal constitutional infection now agree that the consecutive diseases are altogether different from the sequelæ of syphilis, with which they have nothing in common. The gonorrhœal virus is a *contagium fixum*, its vehicle is the secretion of the diseased mucous membrane, and it is

only by contact of this secretion with a mucous membrane susceptible of infection that the complaint can be transmitted from one person to another, or from the mucous membranes of one organ to that of another in the same individual. As in other infectious disorders, between the time of infection and that of the outbreak of the disease there is a certain interval, known as the period of incubation. The length of this period is from three to eight days. The appearance of a gonorrhœa as early as twenty-four hours after an impure coitus, or as late as three or four weeks afterward (*Simon*), if it occur at all, is to be regarded as a rare exception. Credulous persons will hit upon cases where the period of incubation has lasted longer still. Every physician who has had much to do with venereal patients, especially with patients from the better classes of society, will have found out that it is much easier for a patient to confess to excesses perpetrated six or eight weeks ago than to those of which he has been guilty but recently. The more reason he has to be ashamed of himself, so much the more is he disposed to antedate his delinquency. Least of all are married people to be trusted in this respect, and their assurance that "they would just as lief confess to sins of a week's standing as those of six weeks" is not to be relied upon.

Contact of a mucous membrane with gonorrhœal virus does not always result in infection. Indeed, the susceptibility to contagion varies greatly in different individuals. Daily experience teaches us that two men may have intercourse with a woman having virulent fluor albus, one of whom may contract gonorrhœa, while the other may escape. We do not know by what causes this predisposition to gonorrhœal infection is increased or diminished. It cannot be attributed either to the greater or less degree of venereal excitement during coitus, nor to incomplete introduction of the penis, nor to the degree of "acclimatization" of the exposed individual to the person of the other. It is idle to indulge in unsupported theories upon this question. We do not even know why it is that, of all the various mucous membranes of the human body, only that of the urethra, those of the female genitals, the eye, and in some degree those of the rectum, are susceptible of gonorrhœal inflammation, while all others are quite proof against the infection. Even different portions of one mucous membrane exhibit different degrees of susceptibility to gonorrhœal poison. Although the infecting secretion acts first upon the orifice of the urethra, the gonorrhœa is most apt to develop in the fossa navicularis.

Anatomical Appearances.—Opportunities of making *post-mortem* examination of patients with gonorrhœa are rare, and it was long ere we could obtain positive knowledge that the seat of gonorrhœa was the urethra. In recent cases of the disease the mucous membrane

is reddened, injected, swollen, and coated with puriform secretion. It is a matter of importance for the prognosis and treatment of gonorrhœa to know that, in the first and second week of the complaint, the anatomical changes only involve the anterior portion of the urethra—the fossa navicularis—which is very richly endowed with glands; and it is not until afterward that the lesions extend to the membranous and prostatic portions of the urethra. In very severe cases, the inflammation of the mucous membrane is accompanied by inflammation and infiltration of the corpora cavernosa, which not only diminish the calibre of the urethra, but render a uniform enlargement of the penis during erection impossible. Far more rarely submucous abscesses form in severe gonorrhœa, or, what is a much more serious accident, inflammation and suppuration of the prostate occur. The lymphatics of the penis may also become implicated in the inflammation, and sympathetic swellings of the inguinal glands are not unfrequent complications of the disease, although suppuration in such cases is quite exceptional.

The most common complications of gonorrhœa are inflammation of the epididymis and catarrh of the bladder. These affections both make their appearance about the end of the first or second week, that is, at the time when the pars prostatica becomes involved in the inflammation, and when opportunity is afforded for the extension of the inflammation to the vasa deferentia and neck of the bladder.

In chronic gonorrhœa the mucous membrane is swollen and studded here and there with fungous granulations. Its follicles are enlarged and its secretion is more mucous. In many cases, circumscribed spots, and sometimes extensive tracts of the submucous tissue, are hypertrophied, indurated, and firmly attached to the mucous membrane. It is to this that most urethral strictures are due.

Symptoms and Course.—The commencement of a clap is announced by an itching sensation at the mouth of the urethra, which does not amount to pain, accompanied by a scanty secretion of a transparent, clear mucus. At the same time the meatus seems slightly reddened, and its lips are usually agglutinated by the dried secretion, a thin scale of which is also spread over the tip of the glans. An increased desire to pass water then sets in; the patients often have nocturnal pollutions, and during the day suffer from frequent erections, which often induce thoughtless persons to indulge in further excesses. Gradually, generally in the course of a day or two, the sense of itching in the urethra gives place to a burning pain, extending from the meatus to the fossa navicularis. This pain increases, and, during the act of micturition, is extremely severe. The inclination to pass water becomes more frequent than before, so that with each effort a few

drops only are voided, and those with the utmost suffering. The secretion, formerly scanty, tenacious, and transparent, gradually becomes more copious, thicker, and purulent, making yellow, stiff stains upon the patient's linen. The lips of the meatus are red and swollen; the entire penis also, especially the glans, is more or less swollen, and the urethra throughout its entire length is sensitive to pressure. At this period, the prepuce, irritated by the discharge, or else owing to propagation of the irritation, often becomes excoriated and œdematous; so that the product of a balanitis is added to the secretion which flows from the urethra. If the outlet of the prepuce be small, a phymosis is very apt to occur, or paraphymosis, if the patients imprudently retract the prepuce behind the glans. At this stage erections occur with still greater frequence than at the outset of the disease; but the stretching and expansion which the organ undergoes during the process cause the most intense pain to the patient, deprive him of his rest at night, and induce him to resort to the wildest expedients to arrest his suffering. All these symptoms, the painful micturition, the flow of thick, yellowish-green pus, the redness and swelling of the urethra, and the painful priapism, usually continue to increase for a period of about eight or fourteen days. After attaining their acme, the pain upon micturition usually begins to abate, the redness and swelling of the meatus gradually subside, the erections are less frequent and less painful; but at this time the discharge is often more profuse than ever, so that the laity regard this as a favorable sign, and think that the "clap must be made to run" in order to relieve their suffering.

After a lapse of a week or two the discharge gradually diminishes, becomes mucous, and may finally cease altogether in the course of five or six weeks without any treatment whatever, as has been proved by the results of homœopathic practice. Much more frequently, however, there remains a stationary scanty mucous discharge which may last for months and years. During the day, if the intervals between the acts of micturition be long, this secretion glues the lips of the urethra together. When the patient awakens in the morning, a tolerably large drop of it has collected, and runs out between the adherent lips of the meatus as soon as they are separated. The stiff stains upon the linen are now of a more grayish color, although generally there is a small but distinct yellow spot in their middle. A discharge of this kind is called "gleet" or "goutte militaire." If, while it lasts, the patient exposes himself to further exciting cause, the gonorrhœa not unfrequently breaks out again; that is, the pain does not return but the discharge once more becomes abundant and more purulent. Excess in wine or sexual intercourse is the most apt to cause such

relapses, but exposure to cold and over-exertion seem to have a similar effect.

The symptoms and course of gonorrhœa present a good deal of variation. First of all, they vary in the degree and duration of the inflammatory symptoms, and the pain, redness, and swelling of the urethral mucous membrane. A classification of the affection into various species, such as the erysipelatous, synochal, erethetic, and torpid, has been based upon this variation, without, however, any practical advantages being derived from such arrangement. Generally the inflammatory symptoms of a first clap are far more severe than those of the second and third, but there are exceptions to this rule. In severe gonorrhœa, with intense hyperæmia, rupture of small blood-vessels with hæmorrhage often occurs, imparting a reddish or brownish hue to the discharge. Although this hæmorrhage is not at all dangerous, yet the "bloody," or "black," or "Russian clap," has a terrible reputation among the laity. The flexion which the penis undergoes during erection (known as *chordee*) is of greater moment. These flexions result from a loss of elasticity on the part of the inflamed portion of the corpus cavernosum, which prevents it from participating in the enlargement of the penis. It sometimes happens that the inflamed portion of the corpus cavernosum undergoes permanent atrophy, and that thenceforth the penis when erect is distorted. Sometimes, too, when the atrophy extends entirely through the corpus cavernosum at one point, ever afterward erection is only practicable from the root of the organ up to that point. Among the less serious accidents which may occur during gonorrhœa, is the formation of small abscesses about the urethra. Severe pain, increased by pressure, and a hard circumscribed swelling about the urethra, are the characteristic signs of the formation of such abscesses. They nearly always run a favorable course, whether they perforate externally or into the urethra. A much more dangerous but also a more rare complication of gonorrhœa is inflammation of the prostate. We may infer that the prostate is the seat of hyperæmic swelling, from the unpleasant sense of pressure in the perinæum of which the majority of patients with gonorrhœa complain, as well as from the enlargement and induration of the prostate so often seen in old persons, chiefly in those who have had gonorrhœa in their youth. If the prostate be much inflamed, there is an extremely unpleasant sense of pressure, or a dragging, throbbing pain in the perinæum, extending into the bladder and rectum, which is greatly aggravated upon the passage of the urine or the fæces. Both in the perinæum and through the rectum we can feel a tumor of varying size. Micturition becomes more and more difficult, and the dysuria may increase to absolute retention of urine. If the inflammation go on into suppuration, an ab-

scess forms, which may point either externally or else inwardly, giving rise to symptoms of the most serious and varied character, for further description of which we refer to the text-books of surgery. Gonorrhœal lymphangitis and lymphadenitis present no extraordinary features. Resolution is the more frequent termination of a gonorrhœal bubo. Suppuration of such a bubo indeed is so rare an occurrence, that it always should excite suspicion of the existence of a glandular chancre. Finally, gonorrhœal orchitis, the most common of all the complications of this disease, is undoubtedly the result of propagation of the inflammation from the urethra to the seminal vesicles and vas deferens. At first the pain in the cord, testicle, and epididymis is not very severe, the patient complaining rather of a sense of weight in the testicle. Soon, however, the pain augments, and the epididymis, which is the chief seat of the inflammation, becomes very sensitive to the slightest touch. To the hard irregular tumor formed by the inflamed epididymis there is soon added an effusion into the tunica propria testis, so that in a few days the testis may become as large as a goose-egg or fist, or even larger. The enlarged testicle is less movable than before, as the thickened and indurated spermatic cord has become less flexible than when healthy. The greater the effusion into the tunica vaginalis becomes, so much the more does the tenderness, upon pressure of the swollen organ, concentrate itself at the point where the epididymis is situated. Generally the disease terminates in resolution, but a slight induration of the epididymis almost always remains for some time, or even during life, occasionally proving an object of groundless hypochondriacal anxiety to the patient. Sometimes, especially in persons suffering from varicocele, the gonorrhœal orchitis recurs several times. The discharge from the urethra almost invariably subsides during the continuance of the inflammation, but as invariably reappears when the orchitis abates. Far more rarely, the affection terminates in suppuration, tuberculous degeneration, or induration.

For some time the term gonorrhœal metastasis has been applied to a variety of morbid processes affecting individuals who had had gonorrhœa. In the majority of cases, it cannot be proved that there is any relation of cause and effect between these affections and the disease in question. Gonorrhœal ophthalmia and the well-known inflammation of the joints known as gonorrhœal rheumatism, or gonorrhœal gout, are the only ones of these maladies in any sense of the word entitled to such name. The former proceeds from a direct transfer of the virus to the conjunctiva, and is one of the most dreadful consequences of the disease. I have seen an instance in which a married man, having contracted a clap, abstained from all sexual intercourse

with his wife, but communicated a gonorrhœal ophthalmia to her and to his child, whereby both mother and child lost their sight, the man escaping. That the inflammation of the joints above referred to actually stands in genetic relation to gonorrhœa may be inferred from the fact that it occurs in individuals who have not been exposed to any other cause of disease; that it attacks patients who have never suffered from such symptoms before, and who never have them afterward, and that it sometimes recurs with every gonorrhœa which the patient contracts, and disappears as the gonorrhœa subsides. This arthritis has no particular effect upon the progress of the main disease, nor do the anatomical lesions of the affected joint, or the symptoms and termination of the affection, present any peculiarity. The seat of gonorrhœal arthritis is almost always in the knee, more rarely the foot or hip-joint, never in the joints of the upper extremity.

Treatment.—The only reliable prophylactic measure which I can recommend is the avoidance of all danger of infection. I do not feel called upon to make further suggestions for the benefit of dissolute men, who merely desire to continue their irregularities unpunished.

We shall spare ourselves any detailed enumeration of the various methods and means employed in the treatment of gonorrhœa, and confine ourselves to a description of the most important and desirable ones.

The best therapeutic results are to be obtained in a perfectly recent gonorrhœa before the symptoms have become severe, as it then generally can be cured in a few days. In order that the number of tractable cases of this kind may be increased—for it is only now and then that we see one—we should make all our patients aware that the disease is continually increasing in extent and violence, so that each day of delay only makes it worse. Such opinions, delivered by physicians who have the confidence of that portion of the public among whom gonorrhœa is most common, have marked effect. It is scarcely credible how coolly and with what cynicism these people talk of their debaucheries and their consequences, what an extensive knowledge of the subject is shown by some of the laity, and how much one can learn from them. For instance, in Magdeburg, where the innumerable commercial travellers of the various mercantile houses meet annually at the hotels, report is always made as to whose pox has relapsed, and who remained cured, and what injections have answered the best for gonorrhœa, etc. Only a short time after I had begun to prescribe injections of tannin in recent claps, where the symptoms were still trifling (and I had been very successful with this treatment), the number of recent gonorrhœas which sought my aid multiplied considerably. I usually ordered three powders, each of which contained half a drachm

of tannin. One of these was to be dissolved in a half pint of red wine, and the solution was to be used as an injection. If the result was unsatisfactory, the two other powders were to be put into the same quantity of wine, and the injection to be continued with this doubly-strong solution. It has repeatedly happened to me that a patient has pulled one of these very powders out of his pocket which I was about to prescribe for him, and which he had obtained from a friend, and has asked whether he might try that first. In order that these injections may be of service, we must give them once or twice ourselves, or let a skilled assistant give them. If we neglect this precaution, it often happens that the liquid does not enter the urethra at all, or perhaps is merely thrown under the prepuce, and flows back past the syringe. Gonorrhœa syringes must be so small that they will not hold more liquid than the urethra is capable of containing. It is then unnecessary to compress the posterior end of the canal. The best plan is to have a supply of suitable syringes at some particular instrument-maker's, and to give them some unobjectionable name, since many patients, feeling too much embarrassed to ask for a "penis-syringe," get an "ear-syringe," or other unsuitable article instead. I have cut short a large number of recent virulent gonorrhœas in two or three days by injection of tannin. Even where the disease is not quite recent, but where the inflammation is not very violent, I have often used the tannin, and obtained excellent results, although the cure was less rapid. I have no idea of claiming especial qualities and merits for tannin; but I have employed this article much more often than nitrate of silver, sulphate of zinc, sugar of lead, and other astringents. I have never had occasion to prescribe the strong solution of nitrate of silver (gr. x—xv. to ℥ j), for its effects can hardly be better than those obtained from tannin, and since even its introducers admit that it sometimes causes severe and even violent symptoms, such as I have never seen in my treatment. When the inflammation is very severe, it is well to wait for its abatement before resorting to injections. A restricted but not over-restricted diet and a strong cathartic of calomel and jalap are also advisable in such cases. The customary prescription of milk of almonds makes the patient unnecessarily conspicuous, without being of the slightest benefit to him. When there is extreme tenderness to pressure along the urethra, but only then, we should apply from ten to fifteen leeches to the perinæum. Cold applications are also serviceable; but, to do good, they must be kept up continuously. Cold compresses must be changed industriously, or the patient must be allowed to sit for a long time in a sitz-bath. Compresses which are allowed to remain until they grow warm, and sitz-baths continued for a short period only, merely increase the tendency to erection, and aggravate

the pain. After the inflammation has subsided somewhat, we should proceed to the use of injections of tannin in these cases also. The fact that their action is less certain in this stage of the disease is probably because, when of long standing, the inflammation is no longer confined to the more accessible anterior portions of the urethra, but has spread into the posterior regions, where an injection can reach it less easily. In tedious cases, in which injections of tannin are more apt to fail, I have often made use of the other astringents above mentioned, without, however, obtaining any better success. When astringent injections remain without effect, it is time to have recourse to cubebs and balsam of copaiba. There is no doubt but that these articles are also efficacious in the earlier stages of the disease, and that many a clap has been "aborted" by their use in free doses. If, however, we can succeed without them, it is better to do so, rather than to subject the stomach and intestine to the action of these noxious substances. It not unfrequently happens that protracted gastric and intestinal catarrhs result from the abuse of cubebs and balsam of copaiba. Moreover, the abortive action of these remedies, according to my experience, is often merely temporary—much oftener than is that of injections—so that patients, who have supposed themselves well of their disease, in a few days have as free a discharge again as they had before using the medicine. The idea, that strictures are more liable to occur under the use of injections than where gonorrhœa is treated by internal medication, is based upon an error. It is true that formerly a great many patients who were treated by injections had strictures afterward; but the reason of this was, that injections were only resorted to in very chronic cases of the disease, while cubebs and copaiba were given in the more recent ones.

The long duration of a gonorrhœa is the most frequent cause of strictures. The early use of injections, and consequent speedy stoppage of the blennorrhœa, is the best preventive of their occurrence; just as permanent thickening of the conjunctiva is best averted by the early and active applications of astringents. Where cubebs and copaiba are used, it should be in full doses, which, however, must not be kept up too long; that is, not longer than three or four days after the discharge has ceased. Large doses are relatively better borne than is the long-continued administration of smaller ones, while the latter do not accomplish the object better, even when kept up for weeks. Cubebs alone can be taken very well, if finely powdered and stirred up in soda-water. Four or five heaped teaspoonfuls of the powder may be given in the day. Balsam of copaiba is best prescribed in gelatine capsules, four, six, or eight of which may be taken daily. Should we wish to combine the two articles, we recommend the pills of cubebs,

℥ ss., bals. copaibæ ʒ ij, cera alba q. s. u. f. pil. 120. About three boxes of such pills will be required; ten of the pills to be taken at first three times a day, and afterward four times a day. The gelatine capsules, containing extract of cubebs and balsam of copaiba in combination, are also to be recommended. If there should be a diarrhœa, with severe scalding pain about the anus, or if an eruption—spots of roseola—appear upon the face and body (which is not uncommon), the use of the medicine must be suspended.

The results of treatment in a recent gonorrhœa are most satisfactory; that of an old inveterate gleet, however, is quite the reverse, and the longer it has lasted so much the worse will the prognosis be. Before all else, we must satisfy ourselves, by the introduction of a catheter, as to the existence or non-existence of an urethral stricture. When there is such a stricture, the inflammation often lurks just behind it, and no benefit is to be expected from the use of injections which cannot well reach the affected point, until the stricture has been relieved by the use of bougies. Where there is no stricture, or where the existing stricture has been dilated, we should inject a strong solution of tannin or of nitrate of silver, and, when this fails, should introduce a sound, smeared with an ointment of lunar caustic, into the urethra. Guthrie's unguent. opthalmicum magicum may also be employed (arg. nit. gr. ij—g. x, ung. cetacei ʒ j, liq. plumb. gtt. xv.)

The hæmorrhage which sometimes occurs during gonorrhœa scarcely ever requires particular attention. If it be exceptionally profuse, we may apply cold, or endeavor to stanch the bleeding by compression of the point whence it proceeds. In order to prevent the painful erections which occur chiefly during the night, the patient should eat but very little food toward evening, and, above all, should avoid drinking. If, nevertheless, his rest be disturbed, and if the customary popular remedies fail of effect (such as walking barefoot about the room or jumping from a chair), we may give a Dover's powder in the evening. I have no personal experience of the effect of lupuline, which has also been recommended. If chordee develop, apply leeches (but rather to the perinæum than to the penis), and give an opiate at night. Abscesses forming in the urethra require poultices, and the prompt puncture of any fluctuating point. Upon the first signs of inflammation of the prostate, apply a large number of leeches to the perinæum, and, after the leeches have fallen off, encourage the bites to bleed, by the application of hot stupes. Sometimes the leeching has to be repeated. Internally give calomel with opium, in divided doses. Whether this last prescription really be of service I do not venture to say. The catheter is never to be applied without the utmost caution, and in some cases it must be laid aside altogether for a while. If the

retention of urine be absolute, puncture of the bladder may be necessary. Fluctuating abscesses of the perinæum are to be opened promptly. For further information regarding inflammation of the prostate, we refer to the books of surgery.

The lymphangitis and lymphadenitis usually subside quickly under simple rest in bed. If, after the gonorrhœa has entirely abated, there still remain a swelling in the inguinal region, we should resort to compression, a practice which has often and very improperly been applied to syphilitic buboes. If the patient be walking about, let him wear a hernia truss with a large pad. If he be confined to his bed, a heavy bag of shot, which, not being quite full, yields and makes a uniform pressure over the tumor, is the best means of compression. In order to avert gonorrhœal orchitis, it is desirable to cause all patients with gonorrhœa to wear a suspensory bandage, to which suitable appliances may be attached to save their linen from becoming soiled. This latter precaution is necessary, as the envelopment of the penis in rags, made fast with bandages, does harm. We should select and apply the suspensory ourselves, so as to make sure that it does not "bind" anywhere. Badly-fitting ones, of course, do more harm than good. Upon the first sign of orchitis the patient must betake himself to bed, and there remain, with a wedge-shaped cushion between his thighs, upon which the scrotum is to be so arranged that there shall not be the slightest strain upon the spermatic cord. Besides this, a large number of leeches should be applied to the cord, the after-bleeding from which should be encouraged. The pain almost always subsides after the depletion. When this has been accomplished, hot poultices must be applied night and day to the scrotum, and, should there be any fresh attack of pain, the leeching must be repeated. Much benefit has also been ascribed to the internal exhibition of calomel combined with opium in this affection. Compression of the testicle, by means of which abatement of the pain and rapid subsidence of the tumor are sometimes effected, is a procedure which often fails, and which, according to my experience, generally can be dispensed with.

CHAPTER II.

NON-VIRULENT CATARRH OF THE URETHRA.

Simple non-virulent catarrh of the urethra is a somewhat rare affection. Local irritation of the urethra by foreign bodies, irritating injections, sexual excess, especially coitus practised during the menstrual period, are its most usual causes. A symptomatic catarrh also accompanies urethral ulcers, particularly urethral chancres. In other

instances, the inflammation of other organs, as the prostate or bladder, spreads to the urethra.

The symptoms of non-virulent urethral catarrh are swelling and redness at the meatus, painful burning along the urethra, especially during micturition, and the discharge of a scanty mucous secretion. All this usually subsides in a day or two without medical aid. The more intense and protracted catarrh which accompanies a urethral chancre however, is attended by purulent discharge, and may easily be mistaken for a gonorrhœa. We shall have more to say as to the distinction between the two conditions, in treating of urethral chancre.

Avoidance of the causes from which the affection proceeds, and removal of the conditions by which it is kept up, are the sole treatment requisite for this unimportant and mild disease.

Occasionally a simple catarrh of the urethra only shows itself by adhesion of the lips of the urethra in the morning. Then the patient, annoyed at the idea of having a gleet, presses and squeezes the penis till he brings out a little mucous discharge. This may be relieved by warning him against thus irritating his urethra.

DISEASES OF THE SEXUAL ORGANS.

A.—DISEASES OF THE MALE SEXUAL ORGANS.

In this section we shall speak only of spermatorrhœa and impotence, leaving the other diseases of the male sexual organs to surgery.

CHAPTER I.

NOCTURNAL AND DIURNAL POLLUTIONS—SPERMATORRHŒA.

From the occurrence of puberty till the virile power has been lost, most men have occasional nocturnal emissions, without our being able to say that they are a sign of disease. If the emissions are repeated at unusually short intervals, if they come without lascivious dreams, if they are accompanied by incomplete erections, or if they occur during the waking state (diurnal pollutions), the condition is a pathological one.

The persons who consult physicians on account of pollutions are chiefly young men from seventeen to twenty-five years of age. They complain of having emissions of semen one or more nights each week, which weaken them greatly, and that they feel particularly dull and listless the day after an emission. We should not have too much confidence in these patients, or rather we should mistrust that they are concealing some important fact. The first glance at these confused and embarrassed young men usually shows that they have a bad conscience; in many cases we may tell what the trouble is from the mysterious letters in which they request a consultation. It is not generally difficult to obtain from these patients a confession that they have previously masturbated, and some even appear to be relieved by making the acknowledgment. We must not be satisfied with this confession, however, but should carefully seek to find out if they do not still continue the practice. This confession is generally far more difficult for them; but I shall not exaggerate in saying that full two-thirds of the patients

who have consulted me for their pollutions have at last acknowledged that they still masturbated. These persons seek aid because they have been frightened by reading the miserable books which give such overdrawn pictures of the results of onanism. They hope that it will only be necessary to tell of the frequent occurrence of the emissions, and that they may be silent concerning the cause.

The case is somewhat different with a second class of persons who seek medical aid for their pollutions. These also have masturbated during youth, but have subsequently ceased to do so; they also, however, have come across some bad book describing the terrible results of the habit, and they have been greatly terrified, and have become very hypochondriacal. They do not have nocturnal emissions oftener than healthy persons, but each recurrence gives new stimulus to the hypochondriasis; they are considered very dangerous, and the patients imagine they are experiencing the evil effects of which they have read. The letters that such patients write often form a wonderful contrast to their personal appearance. From the account of their case as given in the letter, we may be prepared to meet a deplorable-looking being, instead of whom comes in a hearty, robust man, that it is difficult to recognize as the writer of the letter.

A third class of persons who consult the physician for their pollutions do suffer from general debility, are badly nourished and anæmic. They have never been given to onanism; nor do the emissions occur frequently, but the day after their occurrence the patients feel peculiarly dull and relaxed, and are inclined to refer the cachexia from which they are suffering to their pollutions. It is well known that, in diseased, exhausted persons, the excitability of the nervous system is more apt to be abnormally increased than it is in strong and healthy ones, and that inclination to pollutions, as one symptom of erethism, is more frequently seen in the former than in the latter. We often find that persons, who have never suffered from pollutions while in health, are afflicted with them when attacked by severe disease or during convalescence.

Besides the last class of persons, in whom the pollutions are not the cause but the result of the diseased and exhausted constitution, and in whom this exhaustion with its causes must be the objects of treatment, there are patients in whom the repeated pollutions are the only reasons we can find for a feeling of great debility and an unconquerable lassitude. Such patients are very peculiar: they cannot think acutely, are sad, cannot work; they are cowardly, easily frightened, complain of trembling, noises in the ears, dizziness, neuralgic pain in the back of the head, etc. Their complaints remind us most strikingly of those of hysterical women, and it is perfectly justifiable to designate

this collection of symptoms as *hysteria*. It is difficult to understand why pollutions should have so injurious an effect on the organism in a few persons, while by most they are borne without observable harm. We cannot consider the loss of semen as the cause of the exhaustion and nervous disturbance. The sexual excesses to which young husbands generally give way very rarely have any injurious effect on their health; even if they have daily intercourse, most of them remain just as strong as they were previously while perfectly continent. In such persons the loss of seminal fluid is so very much greater than in those who suffer from occasional pollutions, that injurious results would occur much more frequently in them if the loss of the fluid were the cause of the injury. From the favorable results that I have had of late years in treating this affection, and patients with spermatorrhœa by repeated cauterizations of the caput galinaginis, I believe that the hysterical symptoms occurring in it are exactly analogous to those occurring in women who have erosions of the os uteri; or in other words, that they do not depend on loss of semen, but that morbid irritability of the sexual organs may excite extensive disturbances of innervation in men just as it does in women. When speaking of hysteria I shall explain more in detail that erosions of the os uteri do not necessarily induce hysteria, but that this is only apt to occur where there is a decided predisposition for it. The case is just the same in men who masturbate or who have pollutions or spermatorrhœa from irritation of the genital organs. I must, however, warn my readers against taking it for granted that all men who have hysterical symptoms masturbate or suffer from pollutions or spermatorrhœa. Hysteria does not depend exclusively on affections of the sexual organs, either in men or women.

By *spermatorrhœa*, in the strict sense, is understood a condition where the semen is not regularly ejected during a complete or incomplete erection, but where it is washed out by the urine, or flows out slowly while the bowels are being evacuated. The statements of *Lallemand* and several other authors, concerning the frequency of spermatorrhœa, are exaggerated. Increased excretion of prostatic fluid is often mistaken for spermatorrhœa. In the white, frothy, or transparent viscous fluid which sometimes collects in considerable quantity at the mouth of the urethra after sexual excitement without coitus, there are usually no spermatozoa or they are very few in number. Frequently also the frothy fluid secreted from the urethral mucous membrane during a gleet, or the mucous filaments in the urine when there is catarrh of the bladder, are mistaken for semen. The microscope alone can render the diagnosis positive.

The causes of true spermatorrhœa are obscure. Relaxation or

dilatation of the excretory ducts of the vesiculæ seminales and other morbid but not well-known changes (probably catarrh and erosions), in the caput galinaginis, appear to be at the root of the disease.

Lallemand and others have exaggerated the injurious results as well as the frequency of spermatorrhœa. I knew a railroad agent in Magdeburg who, for at least ten years, lost a considerable quantity of semen with every stool, without any observable bad effect on his general health. He was married, and his wife had several children by him while he was affected with the spermatorrhœa; he also acknowledged that, during his daily trips to Leipzig, he not unfrequently committed sexual excesses. In some persons, it is true, the same symptoms occur that we described when speaking of pollutions.

Treatment is comparatively powerless against a morbidly-increased inclination to pollution. The best results are attained by paying particular attention to the constitution of the patient, and seeking to rectify any thing that is out of order. This may be the reason why ferruginous preparations and the natural chalybeate baths are so popular for pollutions, and why some patients are improved or cured by sea-bathing, others by the cold-water treatment. Cold sitz-baths and washing the genitals with cold water have the reputation of strengthening the sexual organs and arresting pollutions; but these should not be used in the evening, especially just at bedtime, for, if used at that time, they absolutely favor the occurrence of pollutions. Heavy suppers and drinking freely of tea, etc., just before bedtime, should be forbidden. It is improper to prescribe camphor, lupulin, and similar medicines.

Greatly as cauterization of the caput galinaginis, by means of *Lallemand's* porte-caustic, was esteemed for a time, it has since gone out of fashion. When *Lallemand's* work first became known, every practitioner considered it necessary to have one of his instruments, but most of them have been laid on the shelf for years. However, where the emissions of semen are abundant, and the constitution of the patient undermined, and where, by excluding other anomalies, we may regard relaxation or dilatation of the ducts of the vesiculæ seminales, or chronic inflammation in the back part of the urethra, as the probable causes of spermatorrhœa, we may cauterize the caput galinaginis, *lege artis*. In other cases we may confine ourselves to preventing constipation, ordering the genitals to be washed in cold water, and treating any existing complications.

CHAPTER II.

IMPOTENCE AND IRRITABILITY, WITH WEAKNESS OF THE MALE SEXUAL ORGANS.

During the period of manhood, complete and permanent inability of performing coitus successfully is rarely seen. Even some deformities of the penis, loss of one testicle, or disease of both, often will not cause absolute impotence. On the other hand, cases of diminished power and of temporary impotence are very frequent, and it is of the utmost importance for the practitioner to be thoroughly acquainted with the various and peculiar forms under which these states may occur. Unhappy marriages, barrenness, divorces, or perhaps an occasional suicide, may be prevented by an experienced physician, who has the entire confidence of his patient, if he can give him comfort and consolation when consulted concerning impotence. The persons that come to the physician for counsel are chiefly young husbands, filled with despair at the discovery that they cannot cohabit with their wives. Not only sensual women, but all, without exception, feel deeply hurt, and are repelled by the husband whom they may previously have loved dearly, when, after entering the married state, they find that he is impotent. The more inexperienced and innocent they were at the time of marriage, the longer it often is before they find that something is lacking in their husband; but, once knowing this, they infallibly have a feeling of contempt and aversion for him. And it is not the lost pleasure or the fear of remaining childless that brings the young husbands to the physician, but a sense of shame, and the knowledge that they are becoming contemptible and disgusting to their wives. This sad secret is often concealed from the nearest relatives, and confided only to the physician, in whose art and discretion the patient has full confidence.

The next most frequent class of persons that consult the physician for impotence are young men engaged to be married, who, previous to their wedding-day, have experimented with lewd women to see whether they were impotent, and who have not succeeded on that occasion. Impossible and disgusting as this may sound to the student, when he acquires a moderate practice he will often encounter persons who will relate such things to him with perfect unconcern.

Under proper treatment the great majority of these cases terminate well, so that, when the desperate spouses fall into the right hands, in the course of time they almost always become happy husbands and fathers. The most frequent cause of temporary impotence is lack of self-confidence, and a consequent straining of the mind for the success

of the coitus. Erections not only come without the influence of the will, but the ardent desire for them interferes with their occurrence. The more unconcerned the individual, the less attention he pays to the erections, the more certain and permanent they will be when there is sexual excitement. The patients usually volunteer the information that they have powerful and continued erections at times when they are of no use, but have none when there is an opportunity for coitus, or that, if erections occur, they pass off even during the coitus, before the ejaculation has taken place. Even when such patients have regained their self-confidence by one successful coitus, and have then retained their virile power for some time, they often have long relapses of their impotence from a single failure of the act. There are also cases where the virile power of the patients returns perfectly when they have intercourse with their wives, and they can even visit them at very short intervals; but it always fails if they make the attempt with some other women with whom they have not previously had connection.

The most frequent cause of this diminished power is onanism; sexual excess or repeated pollutions far more rarely cause it. But the diminished power of the onanist is usually first increased to temporary impotence by reading popular and medical treatises on the results of masturbation. In those writings the loss of manhood is described as the inevitable result of onanism, and the readers are thus robbed of all self-confidence. If both the depressing effect of onanism and the despondency from reading these papers act on the same person, the first attempt at coitus almost always fails. But, as we have already said, the effect of this first failure is to induce subsequent ones for a long time. Other persons, not debilitated by onanism, and undertaking coitus with perfect confidence, fail in the act from being intoxicated at the time; but even such persons may become temporarily impotent from the disturbance of self-confidence by the failure, and from paying too much attention to the success of the next attempt, for fear it also will fail.

In still other cases the only causes for the failure of the first coitus are excessive excitement and a certain embarrassment and anxiety. Such persons have often led an unusually chaste life, and, with a rare innocence after marriage, they have attempted coition, being perfectly ignorant of the process. In the first weeks of their married life they are greatly depressed and troubled by their sad experiences. If we meet them a few years subsequently, when they have healthy, blooming children, they laugh freely over the mishaps of their honey-moon.

Besides the numerous cases of deficient power and temporary impotence under the above classes, we must mention the rarer cases,

where the impotence depends on actual functional debility of the sexual organs during the age of manhood. They are distinguished from the above by the fact that, during sexual excitement, even when there is no psychical impediment, there are no erections; nor do these occur, as they usually do even in children, without a feeling of desire, when the patient wakes up with a full bladder. Occasionally examination of the genitals in such cases shows something abnormal: the testicles are atrophied, small, and soft; the scrotum hangs relaxed, or else the penis is flabby and cool, or very small and hard. In other cases the most careful examination of the genitals shows nothing abnormal. Some years since, a farmer consulted me for his impotence. As he was somewhat over thirty years old, and a strong, muscular man, without excess of fat, and as I found nothing wrong about his genitals, the penis being well-developed, the testicles large and hard, I considered it certain that his impotence was of the first variety, and consequently gave a good prognosis. But the case turned out differently from what I had expected; and, after being married a year, the man was divorced, the impotence having continued all this time. This second form of impotence must also remain unexplained until our knowledge of physiology and pathological anatomy advance beyond their present state.

We shall pass over the cases where congenital malformations, castration, or other imperfections, are the cause of the impotence, as well as those where it is only one symptom of general debility in exhausting diseases, especially in diabetes, and shall only add a few words concerning *irritability with weakness* of the male sexual organs. Some authors include the form of impotence first described under the head of irritable weakness, and, in fact, the persons there described are on the one hand irritable, as they are very readily excited by contact with women or other causes; and, on the other hand, they are weak, for the erections are not strong and do not last long. But by irritability with weakness, in the strict sense, we understand a condition where, during sexual excitement, the ejaculation occurs before actual copulation has taken place, or even before the erection is perfect. This weakness also occurs chiefly among persons who have previously masturbated. As long as strong erections still take place occasionally, the prognosis in these cases is almost always good. Even when, from masturbation, this affection has lasted for a long time before marriage, and continues during the first weeks of wedded life, it generally disappears if the mode of life be regulated, and the sexual desire be moderately gratified, but not artificially excited.

In the first form of impotence the causal indications require chiefly a psychical treatment. It is often enough to tell the patient of the

happy results in similar cases that we have treated, and to assure him that, according to all experience, his disease is unimportant and only temporary. Others are cured by having coitus forbidden. The nonchalance that they thus acquire during sexual excitement and the inattention to the strength and duration of the erections render cohabitation possible, and they have the first successful coitus during the time it was forbidden, while previously it had always failed. For ignorant persons we may order some harmless substance, and promise the best results from it, but, at the same time, forbid coitus for a season. In such cases the patients will often come back in a few days and confess deploringly that they could not abide by the restriction. We should particularly warn all persons, suffering from impotence, against artificial excitement, especially against fingering and rubbing the genitals, and thus attempting to excite erections; we should represent to them most earnestly and continuously both the injurious effects and the indecency of such a procedure. All the so-called aphrodisiacs are useless and injurious. Washing the genitals with cold water, cold hip-baths, and cold douches, occasionally appear beneficial, and we should employ these remedies in the second form of the affection also. In some cases of impotence, and particularly in cases of irritability with weakness, cauterization of the prostatic portion of the urethra, by means of *Lallemand's* porte-caustic, has been remarkably beneficial. Probably in these cases the disease depended on spermatorrhœa due to relaxation and dilatation of the ducts of the vesiculæ seminales; occasionally, also, the operation may have had a favorable psychical influence. In the latter cases, reading *Lallemand's* book would greatly aid the cure, for there the result of the cauterization is pictured in such glowing colors that the description must restore courage to the most faint-hearted. Recently some electro-therapeutists have strongly recommended electricity for impotence; and this treatment (besides which these men, according to their own accounts, advise the impotent husbands to refrain from seeing their wives, and to try coitus with lewd women [!]) is said to be very beneficial. In men of full virile power I have often induced erections by faradisation of the inner surface of the thigh, but, where there was impotence even after using electricity for weeks, I have seen no results worth mentioning. But, as my own observations have not been very numerous, I will not pronounce decidedly on the subject, and shall simply give a short account of the plan of treatment advised by *Benedikt* and *Schultz*. According to *Benedikt* we should place the copper pole of a constant battery over the lumbar vertebræ, and pass the zinc pole forty or fifty times in the direction of the spermatic cord, then transversely over the different zones of the upper and lower sur-

face of the thighs, and then lengthwise in the perineum. Such a sitting should last two or three minutes. Moreover, about three times a fortnight the copper pole should be applied, by means of a catheter-shaped rheophore, to the vicinity of the ejaculatory duct, and passes should be made with the zinc pole in the direction of the spermatic cords. If there are any particularly insensible places, *Benedikt* uses *Faraday's* galvanic brush, and, if the testicles are peculiarly insensible, he passes a strong current through them. The sittings should take place every day, and be continued for some time, as improvement did not take place for months in some cases.

Schultz, in Vienna, has for a long time used the induced current for pollutions and impotence. Under this treatment the success was very poor; but he claims that it is much greater since he has commenced using a constant current. He places the positive pole over the fifth dorsal vertebra, the negative over the sacrum or to the perinæum. Each sitting lasts from one to three minutes, and they are repeated three or four times a week. *Schultz* employs a battery with twenty or thirty *Daniel's* elements of medium size.

B.—DISEASES OF THE FEMALE SEXUAL ORGANS

SECTION I.

DISEASES OF THE OVARIES.

CHAPTER I.

INFLAMMATION OF THE OVARY, OOPHORITIS [OVARITIS].

Etiology.—The Graafian follicle, the stroma of the ovary, and its serous coating, may become the seat of inflammation. The first two forms occasionally lead to suppuration of the parenchyma, the last almost always leaves thickening of the serous coat and adhesions to neighboring organs.

Parturition, with its sequelæ, most frequently causes oophoritis; but, as we exclude the diseases caused by pregnancy, delivery, or childbed from discussion in this section, we shall here pass by puerperal oophoritis. Next to childbed, the disease appears to begin most frequently at times when the ovary is hyperæmic, and is the seat of a physiological injury from the rupture of a Graafian vesicle. The best-known causes of non-puerperal oophoritis are injurious influences acting on the body at the time of menstruation, such as catching cold, getting the feet wet, coitus during menstruation, etc. One attack of the disease predisposes to another.

Anatomical Appearances.—Ovaritis attacks only one ovary. If the inflammation start from the *follicles*, we find one, or more rarely several, of the Graafian vesicles swollen to the size of a pea or a cherry, filled with a variegated exudation, and their external envelopes reddened by capillary injection. At the same time the ovary is usually but little enlarged; except a slight œdema, the stroma appears normal; the serous covering usually participates in the inflammation. In most cases the disease runs a favorable course; the exudation is

reabsorbed and the follicle atrophies; in other cases it degenerates to a serous cyst, and exceptionally ends in suppuration and formation of an abscess. If the inflammation start from the *stroma* of the ovary, the process is usually limited to a decided hyperæmia, an inflammatory œdema, and a proliferation of the connective tissue, which subsequently leads to thickening and shrinking of the ovary. Suppuration and formation of abscesses, or diffuse destruction of the ovary, rarely occurs in these cases. In non-puerperal ovaritis, *Kiwisch* has only twice seen this rare termination. Inflammation of the peritoneal covering is sometimes primary, sometimes secondary to that of the parenchyma. In recent cases the ovary is usually covered by a scanty fibrinous exudation, which unites it loosely to the neighboring parts, particularly to the broad bands of the uterus and the Fallopian tubes, whose peritoneal covering usually participates in the inflammation. Subsequently firm adhesions, by means of fibrous bands and filaments, readily form between these parts; not unfrequently these closely envelop the ovary and openings of the tubes. Exceptionally, peritoneal ovaritis causes abundant exudation, and encapsulated foci are formed in the pelvis.

Symptoms and Course.—The symptoms of a partial peritonitis in the neighborhood of one of the ovaries are the only signs usually furnished by ovaritis. If the peritoneal covering be not inflamed, the disease almost always remains latent. The ovary lies so deep in the pelvis, and is so completely covered by intestines, that pressure downward from above the symphysis pubis does not affect the ovary, unless the abdominal walls are very much relaxed and distensible. Hence it is important to decide, by vaginal examination, whether the ovary be really the origin of the pain. Occasionally the diseased ovary may be reached through the rectum. The etiology, also, may confirm the diagnosis. If we meet the above symptoms in a patient who has caught cold, or received injury in some other way at the menstrual period, and the menses have suddenly ceased, the presumption that the partial peritonitis starts from the ovary is more tenable than that it depends on disease of some other organ, covered by peritonæum. The symptoms may be modified by an extension of the inflammation to neighboring organs. Uterine blennorrhœa and bloody or bloody serous discharge from the uterus, pain in passing water or fæces, neuralgic pains or numbness in the corresponding extremities may accompany ovaritis, while in other cases they do not occur. Oophoritis is only exceptionally accompanied by fever. The course of the disease varies greatly. In favorable cases the symptoms pass off in a few days, without leaving a trace. Probably the combination of symptoms called colica scortorum (colic of prostitutes) depends on

slight ovaritis, which runs a rapid course, and terminates favorably. At least, in women of suspicious character, I have often seen severe pain in the lower part of the abdomen, which appeared to originate in the ovaries, and was increased by slight pressure, rapidly disappear after the application of leeches. If the disease lasts long; if it causes adhesion of the ovary with neighboring parts, and thickening of the peritoneal covering, the symptoms of ovaritis will frequently recur periodically for a long time, particularly at the menstrual periods. As the opening of a Graafian vesicle, even under normal circumstances, is accompanied by symptoms like those of inflammation, it may be readily understood that, under the above-described normal circumstances, it is often accompanied by those of actual inflammation. I know a lady who was treated over ten years ago by a celebrated gynecologist for severe ovaritis, and who has had returns of her disease several times a year ever since. The copious exudations in peritoneal ovaritis, and the perforation of abscesses in the parenchymatous forms, may cause the above-described encapsulated effusions in the pelvis, or even diffuse peritonitis, with rapid course and fatal results.

Treatment.—In acute ovaritis we should apply ten to fifteen leeches in the inguinal region, or, where it can be done readily, we may apply a smaller number to the neck of the womb. Cataplasms and warm baths aid their action. We should also secure copious evacuations from the bowels; but should avoid drastics, and limit ourselves to giving castor-oil and enemata. Calomel and mercurial ointment are not needed in the treatment of ovaritis. If the disease be protracted, the blood-letting should be repeated occasionally. The systematic use of brine-baths and the internal administration of preparations of iodine and iodine mineral waters also appear beneficial.

CHAPTER II.

OVARIAN CYSTS.

[There are various forms of ovarian cyst.

1. *Hydrops follicularis.* Here one or more Graafian vesicles are transformed into cysts, which is proved by the fact that in young cysts of this kind the ovulum is still to be found. It is supposed that such cysts owe their origin to an abnormal thickness of the walls of the ripe follicles, which, under the influence of the menstrual afflux, swell up to the size of a pea, but do not burst, as physiologically they should do. But the appearance of these cysts before puberty, and even in newly-born children, proves that a mere

indisposition to burst is not the sole cause of their formation, and that a morbid state of the secretion within the follicle may be the real cause. Sometimes these cysts are solitary; sometimes they exist in great numbers. They are generally small, and hence often remain undetected until after death. Exceptionally, a cyst or a cluster of cysts makes a tumor of the size of a fist, very rarely of the size of the head. Such a cyst has thick walls, is smooth within, and contains a yellow, serous liquid. In young cysts traces of fresh or altered blood can be perceived. Communication between several cysts due to atrophy of the intervening tissues is rarely seen.

2. The so-called *cystomata*, or cystoids—that is to say, proliferating cysts—are by far the most common and important form of ovarian tumor. By most new authors the histogenesis of a cystoma is sought in the epithelial components of the ovary. According to *Waldeyer's* graphic account, a cystoma does not originate from the ordinary egg containing a Graafian follicle, but rather from the more rudimentary forms of the epithelial portion of the ovary, from the rounded balls of epithelium, and from the tubular structures, the so-called "*Pflüger's tubes*," which have probably originated at the embryonal period of the organ. Instead, however, of normally developing into Graafian follicles, from the very commencement they have developed into cystomata; or else the follicle is converted into a cystoma by repeated proliferation of the epithelium of its inner surface. As in all mixed tumors in their later periods, there is in a cystoma an intergrowth of epithelial, dermoid, and vesicular structures, out of which the various types of tumor are built. This growth by proliferation of the interior of the cyst the author classifies in two groups, glandular and papillary. In the glandular form sections through the cyst-wall everywhere exhibit small, simple, tubular epithelial pits (almost always cylinder-epithelium) in the substance of the wall, which present the character of a glandular formation. The mouths of these tubes soon become stopped up by tough secretion, and then, almost exactly as other "retention-cysts" form, they become first distended pouches and then small sacs. Upon the interior of the walls of these sacs new depressions form, which in their turn deepen and form pouches and cysts, and so on; thus a honeycomb appearance is produced. In the papillary cystoma the proliferation of the connective tissue of the cyst-wall is the main feature. From their inner surface numerous ramifying, shaggy, highly-vascular vegetations sprout. Sometimes these are circumscribed, growing only within a certain limit; sometimes they increase incredibly, filling the whole sac. There are many intermediate forms.

Many of the smaller, more recent cystomata are of a more solid structure, and upon section show numerous vesicles filled with gelatinous matter. Older tumors, which are often as large as the head, and which sometimes widely distend the whole abdomen, and contain fifty litres or more of liquid, consist mainly of a principal cyst, in which the accessory cysts grow, and, as their walls become thinner, finally burst. Thus the multilocular cysts tend to become unilocular.

The contents of a cystoma are not always the same, but consist generally of a stringy, thick, dirty-brown or yellowish-green liquid, of specific gravity 1018 to 1024. More rarely, it is a thin liquid or a stiff jelly. According to *E. Eichwald*, the chemical contents consist of two series of organic substances : 1. Mucous matter, derived from direct conversion of the cell-protoplasm of the epithelium. Mucin is gradually converted into what is known as colloid matter, and further is changed into *mucopeptone* (*Eichwald*), a substance analogous to the *albuminpeptone* of albumen. 2. Albumen appears either free or combined with soda. The latter is converted into *paralbumin* (which then no longer coagulates upon heating), and further into *metalbumin*, which will not precipitate with nitric acid. Finally it becomes *albuminpeptone*, the last of the series. *Waldeyer* considers paralbumin and metalbumin as of value in a diagnostic point of view. The former is never wanting in ovarian cysts, and never present in ascites. A more convenient test of the liquid obtained from the abdomen by puncture is by the microscope, under which the discovery of cylinder-cells is of the greatest importance. Colloid globes (degenerate, swollen cells), blood, pigment, and cholesterin-crystals are often found. Wandering cells are not found unless the cyst-wall suppurates, which, however, is not very uncommon. The liquid of ascites is usually of a clear, light-yellow color, of specific gravity 1010 to 1015, and contains amœboid corpuscles and pavement-epithelium ; and upon standing exposed to the air, if any blood has become mingled with it during the puncture, it often, after a lapse of twelve or twenty-four hours, throws down a clot of fibrin, which never occurs in ovarian liquid. If a viscid substance of a somewhat dark color and a specific gravity of about 1018 or 1024 be drawn from an abdominal puncture, it is hardly necessary to seek any further proof that ovarian cyst is its source.

Ovarian cysts are attached by a pedicle, which is sometimes long and narrow, sometimes short and thick, and which includes the ovarian ligament, the tube, and the broad ligament of the womb. Sometimes the seat of the tumor is upon the side of the womb itself. The main cyst-wall embraces the entire tumor, and in the

larger cystomata adhesions more or less extensive to the neighboring serous surfaces are often found—a matter of great importance in a question of ovariotomy.

Regarding the causes of ovarian cysts little that is trustworthy is known. In exceptional instances such growths are met with even in childhood, and it is probable that many which are not detected until adult age have existed during youth. Of three hundred and forty-eight cases observed by *Lee*, *West*, *Scanzoni*, and *Schroeder*, ninety-seven were prior to the thirtieth year, one hundred and forty-five between the thirtieth and fortieth years, seventy between the fortieth and fiftieth years, thirty-one between fifty and sixty, and two between seventy and eighty. Both ovaries are very often found diseased (fifty out of ninety-nine times, *Scanzoni*); but the changes in the less affected ovary are usually stationary, and both ovaries are seldom found simultaneously in such a state of cystic degeneration as to form large tumors, and it is equally rare for the extirpation of one ovary to be followed by the growth of a tumor upon the other.

3. Dermoid cystomata are always congenital. From the sketch given by *His* and *Waldeyer* of the embryonal development of the so-called axischord, from which the rudiments of the genitals develop, it will be seen that in this part, in which the different elements of the embryonal rudiments grow into one another, and in which especial germinal laminæ are no longer distinguishable, elements of skin, fat, nerves, muscle, and bone might stray into the sexual sphere. Dermoid cysts do not usually become developed until after puberty; they are rare during childhood. As a rule, they are relatively small (about the size of an apple or fist), but sometimes grow as large as the head. The structure of the interior wall of the cyst closely resembles that of the skin. There is an epidermic covering, beneath which is a layer of connective tissue resembling the cutis, and in which irregularly-arranged papillæ can sometimes be found. A layer of fat (panniculus adiposus) is very constantly found beneath the cutis. Sometimes deposits of true bone are found in the connective tissue, which may even contain rudimentary teeth embedded in alveolar processes. More rarely a growth of gray nerve-substance or of transverse striped muscular fibres is made out. The wall of the cyst, like the skin, contains sebaceous hair-follicles, sometimes even sweat-glands; and the cavity of the cyst is filled with the products of these organs, a mixture of epidermic cells, sebaceous secretion, masses of fat and cholesterin, and of hair either still growing or cast off and rolled into a ball.]

Symptoms and Course.—In some few cases the symptoms ascribed to ovaritis in the last chapter precede hydrops ovarii. More frequently there are no premonitory symptoms, and even the cysts themselves excite no symptoms as long as they are small and do not press against any neighboring organ. It depends on the position of the cyst whether it will cause trouble on attaining some size, and as to what the nature and amount of this trouble will be. Cysts of even moderate size, situated behind the uterus in Douglas's cul-de-sac, and pressing the uterus against the bladder, may cause severe urinary difficulty ; this may be either strangury or dysuria, according to the part of the bladder pressed upon. Defecation may also be hindered by small tumors ; and pressure on the nerves running along the posterior wall of the pelvis may cause pain in the small of the back, or signs of pressure on the nerves of the extremities ; these are sometimes pains, sometimes a feeling of numbness. Lastly, œdema and varices of the lower extremities sometimes result from pressure of an ovarian cyst on the venous trunks in the pelvis. Besides the above symptoms, there are sometimes changes in the breasts, particularly swelling, discoloration of the areola, and even secretion of colostrum. Occasionally, too, there is sympathetic vomiting, and a general disturbance like that which takes place at the commencement of pregnancy.

As the tumor grows and rises out of the pelvis, the symptoms of pressure on the pelvic organs usually subside. Many patients then feel quite well, and the disease can only be recognized by examination, of which we shall speak hereafter. In some cases, however, the inconveniences continue. For instance, in spite of the enlargement of the other parts, the wedge-shaped, thin portion of the cyst may extend far down into the pelvis, and continue to cause the symptoms of pressure on the pelvic organs. On the other hand, as it rises, the cyst may stretch the bladder, and thus induce more trouble with the urine.

As the tumor continues to enlarge, the space in the abdomen is gradually lessened ; the movements of the diaphragm are hindered, and it is pressed upward. Then we have the symptoms of compression of the abdominal viscera and of the lower lobes of the lungs. Even a moderate fulness of the stomach, or a slight distention of the intestines by gas, becomes very annoying ; vomiting is easily excited ; the patient becomes short of breath, and bronchial catarrh results from the collateral fluxion to the upper lobes of the lungs. Even the secretion of urine may be interfered with by compression of the kidneys and their vessels. Finally, the general nutrition, which has usually been unimpaired up to this time, suffers

from the various disturbances of the organs engaged in the formation of blood. Anæmia and hydræmia are developed ; the patient loses strength and emaciates ; the menses, which have previously been regular, cease ; even where there is no compression of the veins, the lower extremities become œdematous as a result of the hydræmia ; finally, the patient dies, with the symptoms of general marasmus.

The duration of the disease before its fatal termination varies greatly. It often continues for years ; some patients, however, die much sooner from intercurrent diseases, and not a few as the result of their treatment. The tumor does not usually grow steadily, but increases at intervals. *Scanzoni* has observed cases where the liquid in the cyst increased and decreased periodically. Just before menstruation, the tumor increased in size, as a result of increased secretion from the wall ; when the menses ceased, it diminished again. Some cysts only attain a moderate size, and then remain stationary. In one case that I saw, an ovarian cyst, which developed when the patient was eighteen years old, and rapidly attained a considerable size, remained for twenty years without growing any more. It has not been clearly proved that ovarian cysts can entirely disappear from reabsorption of their contents, but a diminution in size seems to occur occasionally from metamorphosis of the walls, such as ossification.

Among the complications that may arise during the disease, we shall first mention the peritonitis which is so frequent ; this sometimes occurs spontaneously when the cyst grows very rapidly, sometimes it is the result of operation. It is characterized by more or less pain, which is increased by pressure, and by fever. Since it causes adhesion of the ovary to the neighboring parts, it is very important for the prognosis of ovariotomy. Inflammations of the inner surface of the cyst-wall are rarer and more difficult to recognize than inflammation of the external surface. They are usually caused by tapping, and induce a change in the quality of the contents of the cyst. This inflammation is generally painless, and a slight fever is usually its only symptom. Occasionally ovarian cysts burst from excessive distention, or from the action of external forces, and their contents enter the abdominal cavity. This accident may be favorable, or the reverse, according to the quality of the contents. There have been cases where purely serous fluid was evacuated into the abdomen, and was quickly absorbed, and the cyst did not fill again for a long while, and in some cases it never filled. But even in such cases the escape of the fluid into the abdomen is accompanied by severe pain and constitutional disturbance. Where the ruptured cyst

contained a more irritating liquid, or fat, hairs, etc., death resulted from severe peritonitis. In the case above mentioned, rupture of the cyst was caused by concussion of the body twenty years after the formation of the cyst. At first only a moderate peritonitis was induced by the escape into the abdomen of the thick, almost pulpy, contents of the sac, which contained quantities of cholesterin. It seemed as if the fall the woman had was really a piece of good luck. The abdomen was entirely collapsed just after the rupture, and only filled slowly ; but the fulness increased steadily, and, after a few weeks, the abdomen was more distended than ever before. It was evident that the inner wall of the ruptured cyst continued to secrete liquid, which was emptied into the abdomen ; and, besides this, there was peritoneal exudation. Tapping was repeatedly necessary. At the first tapping there were drawn off twenty thousand cubic centimetres of a mixture of the secretion of the cyst and of peritoneal exudation, which weighed fifty-one pounds, being one-third the entire weight of the patient before tapping. Death from exhaustion took place a few weeks after the fourth tapping. Autopsy entirely confirmed the diagnosis made during life. Lastly, instead of rupturing suddenly, ovarian cysts may be gradually opened by inflammation of their walls, and their contents may be evacuated into the abdomen, unless the cyst has previously become adherent to some neighboring organ, in which case they are emptied into it. Such perforations most frequently occur into the rectum, and atrophy of the cyst has been observed in a few cases as a consequence of this.

Not unfrequently ovarian cysts may be recognized by physical examination, even before they have risen out of the pelvis. If situated in Douglas's cul-de-sac, or between the uterus and bladder, or even to the side of the uterus, a distinct, sharply-bounded, and more or less movable tumor may usually be felt through the vagina ; it displaces the uterus in various directions, according to its position. Occasionally, also, we may feel the tumor through the rectum. The more distinctly we can perceive that the tumor does not perfectly follow the movements of the uterus, the more certain is the diagnosis. If the ovarian cyst rises out of the pelvis, it usually forms a round, movable, painless tumor, with a distinct upper border, and there is more or less evident fluctuation. If we move the tumor with one hand, while the other is in the vagina, we find that the uterus only moves with the tumor when the movements are very extensive, and *vice versa*.

[When the tumor is adherent to the uterus, this test, of course, fails ; and then, unless its want of hardness gives the right clue, it

is liable to be mistaken for a fibroma of the womb. It is also difficult sometimes to distinguish between such a growth and a cyst of the ligament, or dropsy of the tube. Parametritic and perimetritic exudations, especially when situated in Douglas's space, can be felt through the vagina as rounded masses; but under conjoined manipulation, it can be ascertained that they are not well-defined ball-like tumors. Moreover, they are accompanied by the symptoms of an inflammation of which they are the product, and after abatement of the inflammation increase no further, but rather tend to diminish. Fæcal masses which are often to be felt through the vagina can be indented like putty by the finger, and disappear after unloading the bowel. As it grows, the tumor rises out of the pelvic cavity, unless held down by adhesions in Douglas's cul-de-sac, in which case violent symptoms arise like those which are presented by the retroflexed gravid womb in a case of incarceratio uteri.

The tumor first appears as a round body in one or other iliac fossa, recognizable by percussion and palpation, sharply marked above, and painless. It may be confounded with sundry other pelvic tumors. From a distended bladder it may be distinguished by the introduction of a catheter. From hæmometra, from interstitial fibroid, and from pregnancy, it may be distinguished by careful palpation per vaginam, showing that the latter tumors are all directly in connection with the vaginal portion of the womb, besides possessing other peculiarities.

As the tumor grows larger it rises in the belly, assuming a middle position, so that when the pedicle is long it is difficult to decide whether the growth springs from the right or left side of the pelvis. Since the bowels are attached to the back of the abdomen, they necessarily yield backward as the tumor mounts in the cavity, so as to lie behind and beside it, although sometimes a coil of bowel lies in front of it. Upon making the patient lie upon her back, the tumor may be grasped more or less distinctly, and fluctuation sooner or later becomes perceptible, notwithstanding the fact that these growths are generally multilocular, because many of the small cysts gradually coalesce so as to form large ones. The tumor may present a uniform surface, or its multilocular character may be disclosed by the distinct ball-like prominences on the abdomen. Its consistence is sometimes uniform, and sometimes elastic and fluctuating in one portion, while at others solid masses may be felt, consisting of papillary growth or extensive proliferation of the cellular tissue.

It is easy to distinguish an ovarian cystoma from corpulence of the abdomen and from meteorism. In such cases the percussion-sound is tympanitic. An ovarian sac with its walls not very tightly

distended, and lying closely in contact with a large part of the abdominal wall, may be so difficult to feel, and its fluctuation is sometimes so marked, that it may be mistaken for an ascites; of course, only with that extreme grade of ascites in which the tympanitic sound of percussion is extinguished even in the very highest part of the belly, and in which therefore the patient cannot by sudden changes of attitude cause dull and tympanitic sounds to be heard alternately upon percussion, a very characteristic mark of ascites of moderate degree. The distinctive signs usually given—that when the patient lies upon the back the abdomen is rounded when there is a cyst and flattened in ascites; and that when there is a cyst, the percussion in the lumbar region will be tympanitic because the bowels are displaced downward and backward, but that in ascites the percussion-sound is dull—deserve attention, but may cause error. The vaginal portion of the cervix often gives no positive information, since the tumor cannot always be touched from below through the vagina. The womb, although at times grown fast to the tumor and dragged up with it into the belly, and at others pushed down, displaced, or bent, now and then presents no change of position.

The state of the lower extremities deserves great attention in the diagnosis. If but little swollen, then the fluctuating abdominal tumor is probably a cyst. In obscure cases exploratory puncture and aspiration, although not quite free from risk, give valuable aid, both by rendering an exact palpation of the tumor practicable, and by furnishing the material for chemical and microscopic tests.

The diagnosis of a dermoid cyst is based chiefly upon its small size and slow growth. Not unfrequently it remains undiscovered during the life of the patient. If, however, it continues to grow, it may lead to perforation into the bladder, intestine, or vagina, or else through the abdominal wall. The discharge of its peculiar contents will then reveal its character. In one or two instances a complete recovery has followed such an evacuation of a dermoid cyst. Malignant degeneration sometimes occurs.]

If the tumor grows, it usually approaches the median line. Very large ovarian cysts, which rise to the costal cartilages on both sides, and fill both sides of the abdomen, can no longer be distinctly bounded and distinguished as separate tumors. The abdomen, which is enormously distended and very tense, is usually more prominent than broad, and changes its shape very little with change of the position of the body. At the same time both inspection and palpation show the irregular shape of the distended abdomen, which is due to the fact that these large tumors do not consist of one tumor, but of a union of several cysts. Wherever the tumor comes in con-

tact with the abdominal wall, percussion is absolutely dull. Since the intestines are pushed upward and to the side by ovarian tumors, the dulness becomes most decided at the prominent parts of the abdomen, where it is full in ordinary ascites ; at the most dependent lateral portions, on the contrary, the tone is less dull and is tympanitic. The uterus may be dislocated in various directions by large ovarian cysts. Hence the result of vaginal examinations varies greatly in different cases. The uterus may be pushed downward so much as to constitute a prolapse ; it may be displaced forward, or the vagina may be elongated, and the uterus elevated, so that the os uteri cannot be reached by the finger.

Treatment.—The treatment of ovarian cysts belongs almost exclusively to surgery. All absorbent remedies are useless, and, as most of them are active, they are injurious. This is particularly true of the preparations of iodine and mercury. Occasionally we may retard the growth of the cyst by laxatives and derivatives ; but, as we can only carry out this treatment for a short time, the effect is only temporary, and it is no advantage to the patient to have the tumor remain stationary for a few weeks, and then continue its growth. But we cannot hope by internal remedies to cause a change in the inner wall of the cyst that shall deprive it of its expansibility. In spite of the slight prospect of benefit from medicinal treatment of ovarian cysts, humanity requires us to give the patients a chance at the renowned baths and springs in Kreuznach, Tolz, etc. [When the tumor threatens life, ovariotomy should be performed. The success of certain specialists of late years is remarkably good. Out of five hundred ovariotomies, *Spencer Wells* has saved three hundred and seventy-two patients, and lost one hundred and twenty-eight.]

CHAPTER III.

COMPLICATED NEOPLASIA AND SOLID TUMORS IN THE OVARY.

From the excessive formation of new connective-tissue cells that accompanies the development of cysts, we have *cystosarcoma*. This forms tumors which rarely attain the size of those described in the last chapter.

Fibroid tumors, unaccompanied by the formation of cysts, rarely develop alone in the ovary, but they have occasionally been observed of very large size. For several years past I have been treating a patient, aged fifty-five years, who has a very hard, nodular fibroid

tumor, as large as a child's head, in the right ovary; it can be moved to the right or left, and is easily rotated on its axis.

Carcinoma of the ovary is of somewhat more frequent occurrence, but it also is rare. Medullary carcinoma is almost the only form that occurs here; scirrhous and colloid cancer of the ovary are very rare. From complication with formation of cysts, carcinoma of the ovary may form immense tumors. This neoplasm almost always extends over large portions of the peritonæum.

Occasionally we may decide with more or less certainty that there is not a simple cyst of the ovary, from the hardness of the tumor, and from the course of the affection differing from that usual to ovarian cysts. In most cases the diagnosis is obscure. If ascites accompany a nodular tumor of the ovary, and we can discover no other cause for it, the chances are that there is cancerous degeneration. In one case of excessive ascites I made the diagnosis of carcinoma of the peritonæum starting from the ovary, even before feeling the nodular tumor of the ovary (which was rendered very perceptible by tapping), by excluding other causes of ascites, and from the account the patient gave of having had pain in the lower part of the right side of the abdomen and right thigh, as well as of varicose veins of the right leg that had preceded the abdominal distention. The treatment of these ovarian tumors is the same as that of ovarian cysts; but the hope of success is even less than it is in the latter disease.

SECTION II.

DISEASES OF THE UTERUS.

CHAPTER I.

CATARRH OF THE UTERUS AND CATARRHAL ULCERS OF THE CERVIX UTERI.

Etiology.—During menstruation the hyperæmia of the uterine mucous membrane is so excessive that the overfilled vessels are ruptured. Before the hyperæmia attains this grade, and when it is diminishing, the mucous secretion from the uterus is increased and changed. This catarrh, which is physiological as it were, becomes pathological if the hyperæmia of the uterine mucous membrane and the change of the secretion last beyond the normal duration of menstruation, or come at a time when no ripened ovum has been detached. Remembering this, we may readily understand why catarrh of the uterus is among the most frequent of diseases, being at most excelled in frequency by catarrh of the stomach, an organ subjected to the same conditions.

The tendency to uterine catarrh varies greatly with the age. It is rare in childhood, when periodical recurrence of physiological congestion of the uterus does not yet exist; during the age for child-bearing it is very frequent; in old age the predisposition is decidedly less.

Among the exciting causes are:

1. Congestion in the vessels of the uterus; in diseases of the heart and lungs, where the return of blood to the right heart is impeded, the hinderance to the flow of blood from the veins of the uterus usually appears as catarrh of the mucous membrane, and is analogous to cyanosis and dropsy of other parts of the body. Still more frequently the obstruction to the escape of blood is nearer the uterus. In many cases, compression of the hypogastric veins by tumors, or, more often, by collections of hardened fæces in the rectum or colon, are the causes of

catarrh of the uterus; we have before said, that some patients suffering from uterine catarrh, dissatisfied with the treatment of their physicians or even of celebrated gynecologists, fall into the hands of charlatans, or use *Morrison's* pills, and for a certain time are benefited by the continued use of these laxatives that are vaunted as panaceas.

2. Many cases of uterine catarrh are caused by direct irritation of the uterus, and, from what was said above, it is evident that any noxious influence acting on the uterus, when in a state of congestion, will prove more injurious than at other times; hence that imprudence during menstruation most readily induces uterine catarrh. The uterus is directly irritated by too frequent or too energetic coitus, by masturbation, or by wearing pessaries, etc. The uterine catarrh accompanying other diseases of the uterus, such as parenchymatous inflammation, neoplasia, etc., and which is usually called symptomatic catarrh, also depends on the direct irritation.

3. Like other catarrhal affections, this also may depend on constitutional disease. Acute catarrh of the uterus occurs in typhus, cholera, variola, and other infectious diseases; chronic catarrh usually accompanies chlorosis, scrofula, and tuberculosis. As we have often said, the connection between the local disturbance and these general diseases is not understood.

4. When we read of the *epidemic appearance* of uterine catarrh, it simply means that, without any known cause, the disease occurs more frequently at one time than at another.

Anatomical Appearances.—Acute catarrh of the uterus is rarely seen *post mortem*. The changes of the mucous membrane of the uterus in acute catarrh do not differ from those of other mucous membranes in the same disease. There are hyperæmia, swelling, succulence, and relaxation of the tissue; the secretion of mucus is diminished at first, but subsequently increased; in the first stages this mucus is clear and deficient in solid constituents; subsequently it becomes yellowish, and contains quantities of young cells.

In chronic catarrh the mucous membrane is more swollen and hypertrophied; it is brownish red or slate gray; the secretion from the cavity of the uterus appears more or less purulent, and is often mixed with blood. The secretion from the cervix uteri, on the other hand, is usually tough, coherent, and forms gelatinous plugs. Where the process has lasted a long while, the structure of the mucous membrane is changed. The ciliated epithelium is replaced by cells without cilia. Part of the glands is destroyed, while others swell up like cysts. In many cases the surface of the uterine cavity, particularly of the posterior wall, is covered with granulations that bleed readily, or with polypoid growths. The swollen and granulated mucous membrane often

projects from the gaping os uteri, particularly when a large speculum is introduced (*Roser's* ectropion of the os uteri).

The *ovula Nabothi* are very frequently found. These are round translucent nodules, from the size of a millet-seed to that of a pea, and filled with liquid, which are seated in the cervical canal and about the os uteri. They are the distended follicles of the portio vaginalis, whose excretory duct is closed, but whose secretion continues.

Both forms of *catarrhal ulcers* that come on other mucous membranes also occur very frequently in catarrh of the uterus, and are very readily observed, as they occur chiefly at the os uteri. Diffuse catarrhal ulcer or catarrhal erosion is most frequently found at the posterior lip of the uterus; it may thence spread to the anterior one. The loss of substance is very superficial, irregular in shape, has a dark-red base, and is usually covered with puriform secretion. Follicular ulcers, which result from the suppuration or rupture of obstructed follicles, form small round losses of substance, which show no inclination to spread laterally. *Granulating ulcers* of the os uteri are almost as frequent accompaniments of uterine catarrh as are the ovules of *Naboth*, simple erosions, or follicular ulcers, and are more important than these. They are distinguished from simple erosions, from which they appear to proceed, by their irregular, granular, readily-bleeding surface. The softness of the granulations prevents our mistaking them for simple erosions occurring over a thick group of ovules of *Naboth*, which have a nodulated granular appearance.

Symptoms and Course.—If we except the virulent form, of which we shall hereafter speak, severe catarrh of the uterus running an acute course is rare. The disease usually begins with symptoms of severe congestion of the pelvic organs, with pains in the sacral and inguinal regions, with a feeling of fulness and weight in the pelvis, often also with dysuria and tenesmus. Pressure on the lower part of the abdomen gives the patient pain, although we cannot feel the uterus. When the disease is mild, these symptoms usually appear without fever; when it is more severe, especially in irritable subjects, they are not unfrequently accompanied by fever. After three or four days, the patients notice a discharge from the genitals; this is at first transparent and somewhat glutinous; it leaves gray spots on the underclothes; subsequently it becomes cloudy, more or less purulent, and leaves yellow spots on the clothes. If we introduce the speculum (a measure which causes great pain if the vagina participate in the disease), we find the portio vaginalis swollen, dark red, and the secretion above described is escaping from the os uteri. While the reaction of the vaginal secretion is acid, this is alkaline. In most cases the pain and any accompanying fever disappear in from eight to fourteen days.

The discharge also generally becomes less copious about this time, or a little later; it loses its purulent appearance, and finally disappears entirely. In other cases these symptoms of acute catarrh are followed by those of chronic catarrh of the uterus.

The latter cases, where *chronic* uterine catarrh is developed from the acute form, are far rarer than those where only the symptoms peculiar to the former were developed at first. In these cases the commencement of the disease can rarely be recognized with certainty. As long as it is not abundant, the patients do not usually attach much importance to the discharge, which is the most prominent, and for a long time the only, symptom of their disease. If asked how long they have had it, they are almost always unable to tell exactly. The daily amount of the discharge varies: sometimes it is slight; in other cases the patient must change the underclothes daily, and must even lay folded napkins under her at night. The dependence of the discharge on catarrh of the uterus may be inferred when glairy plugs are evacuated from time to time, or if we find in the underclothes the gray, stiff spots that this form of secretion leaves. It is uncertain whether a discharge that leaves yellow spots comes from the uterus or vagina. The more readily it chafes the inner surface of the thighs, the more probable that part of it at least is vaginal secretion. Later in the disease it not unfrequently happens that the secretion from the uterine cavity is retained there by swelling of the mucous membrane, and by tough plugs of mucus that obstruct the canal. The retained secretion collects in large quantities, and distends the uterus. Under these circumstances, there are occasionally pains like labor-pains, very painful contractions of the uterus, that are usually called uterine colic. The longer the catarrh lasts, and the more change it has caused in the mucous membrane, the more frequently we see abnormal symptoms accompanying the physiological congestions of the uterine mucous membrane that occur during menstruation. Among these, the most frequent are severe molimina before the occurrence of menstruation, and pain while it lasts, dysmenorrhœa. In other cases there is too much blood lost, or, on the contrary, too little or none at all. Conception is not always prevented; the fact, that occasionally women with very obstinate and excessive catarrh of the uterus conceive, appears to indicate that it is not the uterine catarrh, but the extension of the disease to the oviducts and their closure by the secretion of the mucous membrane that lie at the root of the sterility which is so common in the disease. When women with chronic catarrh of the uterus do conceive they abort easily, and are inclined to placenta prævia. *Veit* believes that both the deep seat of the placenta in the uterus and the sterility common in this disease are due to the circumstance that it is difficult for the ovum to become em-

bedded in the uterine mucous membrane. In the former case the ovule does not become attached near the opening of the ovary, but at a deeper point; in the other case it is not even arrested at the os uteri, but falls through and is lost. The symptoms of uterine catarrh are not much altered by the development of the ovules of *Naboth*, and catarrhal and follicular ulcers at the os uteri. Granulating ulcers, on the contrary, cause pain, and bleed readily during coitus, and are most apt to induce the nervous symptoms of which we shall hereafter speak.

The influence of chronic uterine catarrh on the general health varies greatly. Some women bear even high grades of the complaint well; their nutritive condition, strength, and blooming appearance is all that could be wished for. But some soon emaciate, become dull, relaxed, and pale or dirty-colored, with blue rings around the eyes.

From the anæmia and hydræmia, but particularly from irritation of the nerves of the uterus having a reflex action on other nerve-trunks, we find anomalies of innervation in many patients with chronic uterine catarrh. Most frequently there is general hyperæsthesia; but neuralgic and spasmodic affections, and decided hysteria, not unfrequently accompany chronic uterine catarrh. Proving the frequent association between these so-called "hysterical" symptoms and catarrh and other diseases of the uterus, and carefully studying them up, were certainly steps in advance; but recently there is great tendency to fall into the error of referring all hysteria to uterine disease without due examination, thus neglecting the other causes of hysteria. Since in this disease the whole attention has been paid to the portio vaginalis, and both laity and physicians have almost exclusively given over the treatment to gynecologists, it cannot be denied that many cases are cured which would formerly have remained uncured; but many also remain uncured now, which would formerly have received aid. Hence it is important that every physician should have a certain acquaintance with "the diseases of women," and particularly that he should understand the use of the speculum. If he do not wish to enter on the local treatment, he may turn the case over to a gynecologist; but he should be able to determine whether local treatment is advisable or not. The fact, that the objection women have to the use of the speculum is almost overcome, is due to the specialists. In the upper classes the women and their husbands consider it as a matter of course that, when there is fluor albus, the speculum must be introduced; but among the middle classes also, a physician who has the confidence of his patients will find but little difficulty if he says that it is necessary to make a careful examination with the speculum. It is only by introducing this instrument that we can attain any certainty as to the source of the discharge, and concerning most of the changes of the os uteri above

mentioned. Exploration with the finger alone can, at most, decide that there is swelling of the portio vaginalis, which, as we shall hereafter see, accompanies most cases of uterine catarrh, and that the ovules of *Naboth* are present. Catarrh of the uterus generally runs a very tedious course. The disease may drag on for years, and it often defies all treatment. In the proper place we shall speak of chronic parenchymatous metritis, flexion and closure of the cervical canal, as the frequent results of this disease, which, in other cases, is due to this closure, and is kept up by it.

Treatment.—In the treatment of catarrh of the uterus it is most important to fulfil the causal indications. Grateful as we should be for the labors of recent gynecologists, and brilliant as the results of their treatment of uterine catarrh appear when compared with the results of former treatment, still some of them are not free from the reproach of neglecting the causal indications while attending to the indications from the disease. Where catarrh of the uterus is one of the symptoms due to general venous congestion, dependent on disease of the heart or lungs, very often the causal indications cannot be fulfilled, and the remedies that have a beneficial effect on catarrh of the uterus are not employed for the uterine disease, but for some other disturbance. If the congestion of the uterine mucous membrane be due to habitual constipation, proper treatment of this disease, as previously advised, has the best results. We do not treat catarrh of the rectum, which depends on congestion of the hemorrhoidal veins due to habitual constipation, with local remedies till we have tried whether "sublata causa cessat effectus;" nor should we employ local treatment for catarrh of the uterus till we are satisfied that removal of the existing constipation is not sufficient for the cure. Then the peculiar conditions, of which we shall hereafter speak, will preponderate. After active local treatment, laxative mineral waters are often prescribed as after-treatment; and these do more good than the actual treatment, because they fulfil the causal indications. A woman named *Graff*, living in Thuringen, has an enormous practice among patients with leucorrhœa. They all drink complicated infusions of manna, rhubarb, senna, and other laxative remedies, and many bless the result of this treatment, to which they were driven by the lack of success of all previous treatment. Physicians, who do not pay sufficient attention to the causal indications in treating catarrh of the uterus, deserve the blame for this. Of course, any thing, that could have induced or can keep up irritation of the uterus, must be carefully removed and kept away. Hence the causal indications may require the removal of tumors of the uterus, or the cure of other changes of structure which induce the catarrh of the uterus Where the uterine catarrh depends on constitutional disease, it is not

always possible to remove the cause; but frequently the original disease is so important, or other dangerous results of it are so prominent, that we cannot attend to the uterine catarrh. This is particularly true of tuberculosis.

Finally, it is not always possible to say whether anæmia and chlorosis are the results or cause of this disease. If we think that the sequence in which the symptoms occur and other causes justify us in the latter supposition, we may often obtain the best results from the use of iron, quinine, a moderate amount of wine, and nutritious diet. Moreover, the good result of cold-water treatment, sea-bathing, and different mineral waters in uterine catarrh, is due to the fact that they have fulfilled the causal indication. Any practitioner will bear witness that the constitution suffers in many cases without our being able to discover the cause; and that anomalies of the constitution, which show themselves by a change in the secretion and function of different organs, cannot always be cured by preparations of iron and nourishing diet, even if there are evident coexistent signs of anæmia and hydræmia. Under such circumstances, all we can do is, to change and improve the constitution by placing the patient under the most different circumstances, changing the entire mode of life, and particularly by modifying as much as possible the exchange of tissue by baths and douches, by giving quantities of water with or without the addition of salts, and by other means. Among the anomalies of secretion that occur in the different organs of such patients, catarrh of the uterus is very frequent; and it often disappears very quickly when we succeed in improving the constitution, while it does not yield to exclusively local treatment. I have seen the most surprising results from such treatment in the Greifswalder clinic, where the arrangements to some extent replaced the treatment by mineral waters and baths, and where Professor *Liebermeister*, at that time assistant physician of the medical clinic, kept account of the effect of the changed diet, increased exercise, and copious supply of salty liquids, of the baths and douches, by weighing the body and examining the urine.

The indications from the disease may be far more readily fulfilled in catarrh of the uterus than in catarrh of other organs that are less accessible. The uncertainty of internal remedies for catarrh has been repeatedly mentioned. They may be dispensed with in the treatment of uterine catarrh, and muriate of ammonia (which many physicians consider just as efficacious for bronchial catarrh as for gastric and intestinal catarrh) is not used in uterine catarrh because we have better and more certain remedies for it. I should be entirely misunderstood, if it were supposed that I considered the local treatment of uterine catarrh as superfluous, or underrated its results; in what was said

above, I only intended to show that one indication should not be followed to the neglect of the others. In all cases where the cause of the catarrh cannot be discovered, as is most frequently the case, local treatment must be used; and where it has existed for a long time, and is complicated with ulcers, particularly granulating ulcers, local treatment should be used with that for fulfilling the causal indications. Among the local remedies we shall first mention injections into the vagina. It is not long since these constituted the only local treatment for "leucorrhœa," no matter whether it came from the uterus or vagina. They aid the treatment and are required for cleanliness, although they are of far less use than the procedures to be hereafter mentioned. In acute catarrh we inject lukewarm water; in chronic catarrh, at first lukewarm, and afterward cold water, or solutions of sulphate of zinc, tannin, or alum. [*Thomas*, *Emmet*, and other eminent gynecologists of America prefer to employ vaginal injections of water, of as high a temperature as the patient can comfortably bear, the action of heat upon the tissues being more enduring than that of cold.] Instead of using an enema syringe with a uterine nozzle, it is well to employ a clysopompe (Davison's syringe), so that we may throw in a large amount of liquid without irritating the vagina by frequent introduction of the nozzle. The application of leeches to the os uteri in acute catarrh is indicated when it begins with great severity, and in chronic catarrh when the substance of the uterus participates in the inflammation, or when there is acute exacerbation of the disease with symptoms of severe congestion in the pelvis. *Scanzoni* also recommends them when there are granulating ulcers on the os uteri. We should apply leeches to the os uteri ourselves, or have it done by a nurse skilled in the operation. Gynecologists of the present day are refraining more and more from the use of leeches in the treatment of catarrh of the uterus and ulcers of the os, while formerly they were used far too often. The local application of nitrate of silver, in substance or in strong solution, is by far the most effective treatment of chronic uterine catarrh, and particularly for catarrhal erosions and follicular ulcers of the vaginal portion of the uterus. To prevent the caustic from breaking off in the cervical canal, we should employ sticks of double-annealed nitrate of silver, or have it hardened by the addition of a few grains of nitrate of potash. When thus prepared, we may push it boldly into the cervical canal. If cauterizations with solid nitrate of silver cause hæmorrhage, which is often the case even in simple ulcer, we should use in its place concentrated solutions (one part to two or four of water), which should be poured in through the speculum

rather than used on a brush. I should employ these solutions much oftener, if it were not so difficult to protect the fingers and clothes from being soiled. The application should be repeated once a week or oftener, till the discharge diminishes and the portio vaginalis has regained its normal appearance. The result of this treatment is so striking, that touching the os uteri and its cervical canal, in chronic catarrh of the organ, must be classed among the most gratifying operations in medicine. The pain induced by the cauterization is usually very insignificant, but in some cases it is quite severe. If the nitrate of silver be passed far into the cervical canal, some women will have painful contractions of the uterus, that may continue for hours. Besides nitrate of silver, the remedies most frequently used for catarrhal erosions and follicular ulcers of the os uteri are pyroligneous acid, liquor hydrargyri nitrici, and cuprum aluminatum (lapis divinus). Pyroligneous acid is particularly beneficial where the ulcers have a great tendency to bleed; the liquor hydrargyri nitrici, and still more the lapis divinus, are to be tried when the nitrate of silver has failed. In such cases the actual cautery is a very effective remedy, and the opposition to its use is ascribable to its psychical effect rather than to the pain or danger accompanying it. Pyroligneous acid poured through the speculum is an invaluable remedy for the granulating ulcers of the os uteri that bleed readily. In most cases it arrests the hæmorrhage more certainly than sesquichloride of iron or alum; the latter is applied to the os uteri in substance more readily than in solution. We should only use injections into the cavity of the uterus in cases of absolute necessity, i. e., only when the above treatment fails, and we are forced to believe that the cavity of the uterus chiefly is diseased. In such cases we should use the ordinary solutions of nitrate of silver (℈ ss. to water ℥ j). The effect of these injections is much more severe than that of touching the os uteri with nitrate of silver; they not unfrequently induce severe inflammatory symptoms; where the cervical canal is contracted, they should never be employed.

[Injections into the cavity of the uterus are never to be practised except in cases of the utmost urgency, when other treatment fails, and when the chief seat of disease is known to be within it. For this purpose nitrate of silver (0·5 to 30·0) may be employed, or, when there is a tendency to bleed in consequence of proliferation of the mucous membrane, the liq. ferri chloridi, tincture of iodine, alum, tannin, or carbolic acid. It is well known that the liquid injected may possibly pass through the tubes into the peritonæum; but aside from this danger, the operation not only excites

severe uterine colic, but in many instances has induced a fatal metritis or peritonitis. Hence the operation demands the utmost precaution. A ready escape for the liquid injected must be secured by dilating the cervix by means of sponge-tent, and the quantity of liquid used must be very small. When the womb is tender to the touch, and when we suspect that there is a parenchymatous metritis or other complication, intrauterine injection must not be attempted.]

CHAPTER II.

PARENCHYMATOUS METRITIS—ACUTE AND CHRONIC INFARCTION OF THE UTERUS.

Etiology.—The changes of the substance of the uterus in acute and chronic parenchymatous inflammation rarely go beyond excessive hyperæmia, inflammatory œdema, and proliferation of its connective-tissue elements, in which the muscular elements usually participate but little or not at all. There is rarely suppuration or formation of abscesses. We do not include the puerperal form here.

For the etiology of parenchymatous metritis we may refer to that of the catarrhal form. The injurious influences there mentioned sometimes cause inflammation of the substance of the uterus; at others, of its mucous membrane; but most frequently of both. These influences also cause parenchymatous metritis more readily if they act while the uterus is in a state of physiological congestion. Lastly, the parenchymatous metritis of an unimpregnated uterus must often be regarded as the continuation of a puerperal metritis; or at least a large number of cases date from the period of a confinement or of an abortion.

Anatomical Appearances.—In acute parenchymatous metritis, we find the uterus increased in size, particularly in thickness. It may attain the volume of a hen's egg or larger. The over-filling of the blood-vessels causes its substance to appear more or less dark, and usually irregularly reddened. These changes are most marked in the layers lying next the mucous membrane. Occasionally there are effusions of blood into the parenchyma. The mucous membrane almost always shows the signs of acute catarrh. The serous coat also often participates in the inflammation, and is covered with deposits of fibrin.

In chronic infarction, the uterus is often enlarged to three or four times its normal size; its cavity increases, particularly in the long diameter; its walls may become an inch thick. The hyperæ-

mia which is at first present subsequently disappears, as the vessels are compressed by the neoplastic shrinking connective tissue. Then the substance appears very pale and dry, and becomes denser and harder, often to such an extent that it creaks under the knife. In rare cases we find hyperæmic spots and veins that have become dilated as a result of the obstructed flow of blood. If the vaginal portion be chiefly affected, the os uteri is greatly swollen, and occasionally elongated like a snout. The mucous membrane almost invariably shows the changes described in the previous chapter. On the peritoneal surface we often find firm adhesions to neighboring organs.

Symptoms and Course.—*Acute* parenchymatous metritis begins with a chill more frequently than the catarrhal form does, and is more apt to be accompanied by symptoms of fever in its subsequent course. The pain in the sacral and inguinal regions, the feeling of pressure in the pelvis, the sensitiveness of the lower part of the abdomen, the dysuria and tenesmus, are present in the former as well as in the latter, and almost always attain a higher grade than they do in the simple catarrh. The uterus can rarely be felt above the symphysis pubis, but through the vagina we may usually detect enlargement and tenderness of the inferior segment, and a moderate shortening and thickening of the portio vaginalis. There are also anomalies of menstruation. If, as is commonly the case, the disease begins during menstruation, the bleeding usually ceases suddenly ; if the time for menstruation occurs during the progress of the disease, we either have metrorrhagia (metritis hæmorrhagica), or, as more frequently occurs, there is no bleeding. Except during the period of menstruation, the discharge characteristic of uterine catarrh (the constant companion of parenchymatous metritis) is present. In favorable cases the disease runs its course in from eight to fourteen days, the symptoms subside gradually, and the disease ends in perfect recovery ; in unfavorable cases, chronic infarction remains. There have been some very rare instances where an abscess formed and perforated into the abdomen, and the disease thus terminated fatally.

Except at the menstrual periods, the symptons of *chronic* infarction of the uterus are often not very prominent. Frequently the patient complains only of a feeling of weight in the pelvis and a sensation of "bearing down." The pressure of the enlarged uterus on the rectum and bladder usually causes constipation, as well as a frequent and annoying inclination to go to stool and to urinate. At the commencement of the disease, menstruation is often free and prolonged ; but the more the vessels of the uterus

are compressed by the neoplastic connective tissue, the more difficult menstruation becomes and the scantier the flow. Finally, the menses are often absent for months or years, while the regularly recurring molimina seem to indicate that the ripening and throwing off of the egg takes place at normal periods. In this form of metritis also—which, moreover, is always accompanied by the catarrhal form—the nutritive state of the patient usually suffers after a time, and the hyperæsthesia and other disturbances of innervation, mentioned in a previous chapter, usually develop. On physical examination we may often feel the enlarged uterus through the abdominal walls above the symphysis pubis, particularly if we push it up a little with the finger introduced into the vagina. On vaginal examination, we also discover that the vaginal portion is enlarged, indurated, and more or less painful. On introducing the uterine sound (which should not be employed unless the practitioner is skilled and experienced in its use), the increase in the long diameter may be ascertained. Although not dangerous, the disease is very obstinate and tedious. Even in its advanced stages it cannot be regarded as absolutely incurable ; the decrease after confinement of the uterus, which had been greatly enlarged during pregnancy, renders it not improbable that there may also be a retrocession of the pathologically increased tissues of the uterus. Occasionally improvement and cure of infarction of the uterus have been seen directly after pregnancy, and in such cases it seemed as if, with the involution of the uterus after confinement, there had been at the same time a diminution of the physiologically and pathologically increased tissue.

Treatment.—According to the variety of the exciting cause, the causal indications are fulfilled by the different rules prescribed in the preceding chapters. In many cases the continued use of slight laxatives is very beneficial, particularly the laxative waters of Marienbad, Franzenbad, Kissingen, etc.

The indications from the disease are best answered by the repeated application of leeches (four to six) to the portio vaginalis. Although I have seen a most favorable effect from this treatment, in my own practice and in that of others, where the disease was recent, it has seemed of little use in protracted cases. Theoretically, also, we may expect better results from abstraction of blood while the connective-tissue formation is still new, and the catamenia are plentiful and continued, than when the capillaries have been compressed, the uterus deprived of blood, and the menses have ceased. Before convincing ourselves by personal observation of the brilliant results of abstraction of blood in the first stages of parenchymatous metritis, and finding how well the patients bear the loss, it is usually difficult to make up

our minds to increase the copious loss of blood by applying leeches to the vaginal portion of the uterus every week or two. A more irritating treatment, particularly the continued use of warm douches to the uterus, seems preferable in the later stages of the disease. These douches should be used about ten minutes every day; the water employed should not be over 99° or 103° F. It is also worth while trying the use of Kreuznach and other saline baths, as well as the baths and waters of Krankenheil. The internal administration of iodide of potassium and of bromide of potassium, as recommended by *Simpson*, is also beneficial.

[Acute metritis demands absolute rest in bed, the pelvis being kept somewhat elevated. When, from the tenderness of the belly, we suspect a complication with peritonitis, leeches should be applied to the abdomen and followed by cold compresses. When the inflammation seems limited to the womb, local depletion of that organ should be practised by leeching the vaginal portion of the cervix, unless the virgin condition of the patient forbids it. Scarification by means of *Mayer's lance-knife* has, however, in a great measure supplanted the leech applied to the womb. It has the advantages over leeching that the procedure is quite painless, and that the amount of blood to be drawn can be nicely regulated by making the incisions deeper or shallower. Moreover, many observers believe that the suction of a leech may set up an irritation which tends rather to augment the fluxion toward the affected part than to check it. According to *Schroeder*, scarification should be repeated about every third or fourth day, not more than half an ounce of blood being drawn at one operation as a rule; but if the patient is plethoric and the womb enlarged, an ounce may be taken. Syringing the parts with cold water is a practice which should never follow scarification; for, although the cold produces a momentary contraction of the vessels, a relaxation quickly follows, which brings with it a renewed congestion and thwarts the purpose of the operation.

The demonstration by *Emmet*, of New York, that the majority of so-called ulcerations of the womb are really the effect of laceration of the cervix, is an important advance in the pathology and therapeutics of this subject. He holds that lacerations occur during labor more often than has been hitherto supposed. They occur most frequently in the median line, and then heal readily, and generally escape notice. But if the rent is extensive, and runs in a lateral direction, the weight of the womb tends to force the lips of the wound asunder, so that union by first intention cannot take place. When the woman assumes an erect posture, the flaps diverge still

further, and the mucous membrane of the cervical canal becomes everted and chafes against the vaginal walls. Through the irritation thus set up, the angle of the laceration becomes the seat of an erosion, which may spread over the everted surfaces. Such an ulcer heals with great difficulty, and only by granulation and by the formation of a dense cicatrix. The womb usually suffers subinvolution with all its consequences, and the cicatrices are a fruitful source of neuralgia and other forms of uterine irritability. As long as the fissure remains open, the ulceration is most refractory to all treatment. It is necessary to close the laceration by a surgical operation. This in judicious hands is neither difficult of accomplishment nor dangerous, and yields most satisfactory results.]

CHAPTER III.

PERIMETRITIS AND PARAMETRITIS.

Inflammations of the parts about the uterus occur very frequently just after confinement, and not unfrequently at other times. In the latter case they usually depend on disturbance of the menses. If the inflammation starts from the serous coat of the uterus and its appendages, and the case is one of partial peritonitis, the disease is called *perimetritis;* if, on the contrary, the inflammation is in the subperitoneal connective tissue, it is called *phlegmon periuterina*, or, according to *Virchow*, *parametritis.*

Perimetritis leads to more or less copious exudation on the free surface of the peritonæum. Scanty fibrinous exudations cause adhesions with the neighboring organs. Even large fluid exudations are usually encapsulated by adhesions at their edges. After absorption of the exudation, adhesions to the pelvic organs often remain. In parametritis there is infiltration of the subperitoneal tissue, which is firm from the first. The infiltration may be reabsorbed; but a firm induration often remains as the result of connective-tissue proliferation. In other cases the inflammation goes on to suppuration, and abscesses form, whose contents may perforate into the rectum, vagina, bladder, or abdomen.

It is often difficult to distingnish between perimetritis and parametritis during life; they begin and run their course with more or less severe subjective and objective symptons of fever. The patients complain of pain deep in the pelvis, which is increased by pressure on the lower part of the abdomen. Generally, also, there are symptoms of compression of the pelvic organs, the bladder, rectum,

and, according to my experience, of the nerves along the walls of the pelvis. The presence or absence, and the degree of severity and obstinacy of dysuria and difficult defecation, as well as of the pain extending along the sacral, sciatic, and crural nerves, depend in each case on the seat and amount of the exudation. Where there has been extensive exudation, on examination we may find a tumor of variable size above the pubis. Examination through the vagina or rectum usually shows that the uterus is displaced and firmly wedged in. Intraperitoneal exudations usually fill Douglas's cul-de-sac, and may be readily felt. Subperitoneal infiltrations and abscesses are generally somewhat higher, but they also can mostly be reached by the finger.

[Unless the two conditions should happen to coexist, the results of physical exploration show differences in many respects which can be made out by bimanual palpation. A peritoneal exudation sinks into Douglas's space, as being the lowest level in the abdominal cavity. The cul-de-sac becomes distended and pushed downward. If the exudation has coagulated, or when a liquid exudation is incapsulated so that it cannot yield to the finger, we can feel a more or less voluminous tumor, such as a retrouterine hæmatocele presents, lying behind the uterus and vagina, displacing the one upward and forward, and bulging the other forward and downward. On the other hand, the extraperitoneal exudation of parametritis is found as a rule toward the side of the womb, sometimes on both sides; and through the vagina it feels at first like a resisting infiltration, and afterward like a hard, sharply-defined tumor, which seems to spring from the lateral edges of the womb, the boundary-line between the two being sometimes difficult to determine. In exceptional cases—as when Douglas's cul-de-sac is closed by adhesions—a perimetritic exudation may likewise form in a more lateral region of the pelvic peritonæum, and bulge downward; and conversely, there have been rare cases of parametritic exudation into the areolar tissue, behind and even in front of the uterus and the upper vaginal region. In such instances, when the intra- and extra-peritoneal exudations exist together, a diagnosis becomes extremely difficult.] The disease may continue for weeks, and greatly exhaust the patient by the accompanying fever.

[Extraperitoneal abscesses tend by preference to find an outlet at the surface of the body, especially in the groin, or from the iliac fossa, or after passing under Poupart's ligament to appear upon the front surface of the thigh, or else to perforate backward through the ischiatic foramen and muscles of the buttock. On the other hand, intraperitoneal abscesses open more often into the cavities of

the body, particularly into the intestine and vagina. As a diagnostic hint, we will here mention that pain in a limb, really caused by pressure of an exudation upon a nerve, has now and then been mistaken for a pure sciatica.

Schultze also describes a latent *parametritis posteriori*, of insidious, tedious course, usually due to mechanical causes, and said to produce a shortening of Douglas's folds, and to be a most important factor in the production of anteflexion and anteversion.]

The perforation of subperitoneal abscesses or of intraperitoneal exudations into the intestines or bladder is marked by the sudden decrease in size of the tumor, and by the evacuation of purulent masses with the stools or urine ; perforation into the abdomen causes severe general peritonitis, which quickly proves fatal. Even in favorable cases the patients generally recover slowly. In many of the patients under my observation the neuralgic pains lasted for months.

Treatment.—[Treatment of an acute recent perimetritis demands absolute confinement to the bed, leeching to the groins, the application of ice-bags to the abdomen, mercurial inunction, confinement of the bowels by opium—in short, the treatment of a peritonitis. A recent parametritis calls equally for complete repose, but in other respects the treatment may be less active. The bowels should be gently moved, while cleansing and disinfecting injections should be frequently repeated, to clear away all putrid secretions, which are the common exciting cause of this form of inflammation.]

Local abstraction of blood and the application of cataplasms to the lower part of the abdomen act well in the treatment of recent cases of perimetritis and parametritis. We should continue the use of the latter as long as there is any swelling left, even if there be no pain. · In protracted cases I have found great benefit from the use of warm salt baths, with an addition of mother-liquid, and the internal administration of iodide of iron. At the same time, we must keep up the nutrition and strength by proper diet, and treat any existing fever by antipyretics.

CHAPTER IV.

CONTRACTIONS AND CLOSURES OF THE UTERUS—HÆMOMETRA, HYDROMETRA.

In young persons who develop late, moderate degrees of contraction of the os uteri are quite frequent. They hinder conception,

without rendering it impossible. Moreover, they impede the escape of the menstrual blood, and cause it to collect temporarily in the uterus, and to be expelled by painful contractions (uterine colic). I have frequently known women to menstruate without difficulty after their first confinement, who, during their maidenhood and the first years of married life, had severe uterine colic during menstruation. Great flexions and angular curvatures cause contraction of the cavity of the uterus at the point of flexion, which also impedes conception, and occasions uterine colic during menstruation. Finally, neoplasia, which encroach on the calibre of the uterine cavity and the cervical canal, have the same effect. Perfect closure, atresia of the uterus, is very rare. It is sometimes congenital, sometimes the result of erosions and ulcers, which, in cicatrizing, have caused adhesions. The seat of congenital closure of the uterus is usually at the external orifice, that of the developed form at the internal orifice.

As long as the women continue to menstruate, the menstrual blood collects behind the point of closure, causing *hæmometra*. If the closure does not take place till the menses have ceased, the catarrhal secretion from the mucous membrane occasionally collects in the closed cavity, and distends the uterus. Sometimes this secretion resembles serum or synovia, doubtless because the excessive tension destroys the secreting glands in the mucous membrane, and the latter becomes like a serous membrane. The above state is called *hydrometra*.

In *hæmometra*, which, moreover, depends upon atresia of the vagina oftener than upon atresia of the uterus, the uterus may be gradually distended till it becomes as large as it does in the later months of pregnancy, and the blood contained in it, which is usually black and tarry, may amount to eight or ten pounds. According to the observations of *Scanzoni* and *Veit*, when the distention takes place rapidly, the walls of the uterus are thinned; if it comes on slowly, they are thickened by hypertrophy. In the early stages it is difficult to diagnose hæmometra. During childhood closure of the uterus or vagina is hardly ever discovered. The first morbid symptoms occur about the commencement of puberty. At intervals of four weeks there is severe uterine colic, with a feeling of pressure and weight in the pelvis, and signs of severe congestion of the other pelvic organs, or of perimetritis. At first the patients feel well again after these symptoms have continued a few days; but after four weeks there is a relapse. After a time the intervals are no longer free from pain. The abdomen increases in size; the uterus rises above the symphysis pubis, and may rise as high as the navel.

In the monthly attacks the pains become very severe. The patients emaciate, and may become marasmic; or, if no mode of escape be furnished for the blood, the uterus may rupture, or they may die of peritonitis. The latter disease is particularly apt to occur if the tubes be also filled with blood, and their contents escape into the abdomen. Hæmometra cannot be recognized with certainty, or distinguished from other forms of amenorrhœa or dysmenorrhœa, particularly at its commencement, without a careful local examination. If, at the commencement of puberty, there be uterine colic at regular intervals of four weeks, while there is no escape of blood, and if there be, at the same time, a distention of the abdomen, which periodically increases slightly, we should suspect the development of a hæmometra, and urge an examination. If the hæmometra depend on atresia of the vagina, we find the latter distended to a tense tumor, whose lower end extends into the vestibule. If the external os uteri be closed, the vaginal portion is often entirely obliterated, and the position of the os uteri may not be recognizable. If, on the other hand, the internal os uteri be closed, the vaginal portion may retain its normal length. Besides this, we find the uterus distended to a considerable size; sometimes, but not always, there is fluctuation. The treatment of hæmometra is purely surgical.

It is rarely that a hæmometra terminates favorably without the intervention of skilled assistance. In such exceptional cases the blood finds a way either through the normal passages or else into one of the abdominal cavities. Sometimes menstruation ceases prematurely, or the climacteric arrives, when the symptoms abate, and the bloody contents of the uterus assume the character of hydrometra. The treatment of hæmometra is purely surgical, and consists in opening the sac between two menstrual periods. The mode of operation must necessarily vary greatly according to the circumstances of the case. The procedure is a somewhat dangerous one, and demands attentive study in the books of surgery and gynecology.

Of course, *hydrometra* can only result from acquired closure of the os uteri or vagina, and after the menses have ceased. Mild cases of the disease are seen quite often; severer cases, where the uterus is distended to the size of a head or larger, are rare. The most important symptom of hydrometra is an enlargement of the uterus, which usually occurs gradually, and without exciting attention, while in some few cases it is quite rapid and decided. This may be detected on physical examination, and is often even perceived by the patient. If the walls of the distended uterus be

thinned, there is occasionally distinct fluctuation ; if they be hypertrophied, we do not find this most important sign for distinguishing hydrometra from almost all other uterine tumors. Occasionally there is uterine colic, particularly during severe congestions of the uterus. If the closure be incomplete, these contractions sometimes force out the collected fluid, and, according to *Scanzoni*, occasionally also gases that have formed from them. The treatment of hydrometra consists in surgically making a passage for the liquid, and in attempting, by astringent injections, to limit the secretion of the mucous membrane.

CHAPTER V.

CURVATURES OF THE UTERUS—FLEXIONS AND INFARCTIONS.

Etiology.—[Any abnormal change between the directions in the axis of the neck of the womb and that of its body is called a flexion; but it must be borne in mind that when the bladder is empty the womb is normally bent slightly forward. The bend or angle is nearly always at the internal os. When the deformity is such that the fundus inclines forward, and the curve or angle included between it and the cervix is anterior, we have an anteflexion ; the contrary constitutes a retroflexion. There may also be lateral flexions, but they are rare.]

There are various views regarding the pathogeny of these distortions. Most authors think that the causes of the flexure lie in the uterus itself, and support their view on the fact that, at the point of distortion, the wall of the uterus is always flattened, and its parenchyma loose and reflexed. *Virchow* considers the changes at the point of flexion as secondary symptoms, due to the pressure on the walls of the uterus at this point, and to the anæmia of the parenchyma caused by this pressure. It is his opinion that most distortions of the uterus, particularly anteflexions, are caused by congenital or developed shortening of the ligament of the uterus, and by its consequent fixation on distention of the bladder and rectum. It is most probable that the causes of the flexions are not always the same—that they are sometimes within, sometimes outside of the uterus. [*Schultze* believes that a posterior parametritis of insidious course and somewhat frequent occurrence causes a shortening and rigidity of the fold of Douglas, thereby producing a displacement backward and upward and fixing of the cervix, which is the commonest cause of anteflexion as well as of anteversion.]

Retroflexions, the most frequent form in women who have had

children, are, on the other hand, rare in those who have had none; they almost always date from the time of a confinement or an abortion. If the involution of the uterus go on slowly after its contents have been evacuated, if it remain enlarged and relaxed, the fundus readily sinks down on account of its weight, or is pressed down by the other contents of the abdomen. As the greater part of the uterus remains in the posterior wall after delivery, it is natural that it should most frequently sink backward; but we cannot wonder if there are deviations from this, as they may readily be induced by different positions of the distended intestines in the vicinity, and by other accidental causes. At this period a complete return to the normal state is certainly possible. The bending of the uterus is straightened out when it contracts early. But, if this do not take place soon, the parenchyma, at the point of curvature, becomes anæmic and atrophied from the continued pressure, or the fundus uteri forms some abnormal attachment. In either case we have a permanent anomaly—a distortion in the strict sense. *Scanzoni* mentions, as the most frequent cause, the slow and incomplete involution of the uterus, and, as the most important etiological factors of the retroversion, early marriages, frequent and quickly repeated pregnancies, abortion, artificial delivery, etc.

Anteflexions chiefly occur in those who have had no children. In young persons the most frequent cause seems to be a relaxation of the substance of the uterus by chronic catarrh; in aged persons it is a senile atrophy of the uterus, at the point where these distortions always occur, that is, near the internal os uteri. It is easy to understand that anteflexion should be the more frequent form of distortion in women who have had no children, if we remember that a virgin uterus has normally a slight inclination forward. In this case also, it seems to me, there is no doubt that the flattening of the wall of the uterus, and the atrophy of the tissue at the point of flexion which takes place after a time, are due to the pressure and anæmia.

Besides these flexions, caused by anomalies of the substance of the uterus, there are others which are undoubtedly caused by shortening of the uterine ligaments, as is proved by the cases observed by *Virchow*, where there were flexions without any structural change in the parenchyma. In the same way, distortion may result from fibroid tumors in the anterior or posterior wall of the uterus, from tense adhesions, or from the pressure of tumors. The more securely the lower portion of the uterus is held in place by a rigid vagina, the more readily flexions occur; the less firmly it is held, the more often displacements will occur instead.

Anatomical Appearances.—On autopsy, flexions of the uterus may be readily recognized, as part of the anterior or posterior wall of the body, instead of the fundus, forms the highest part of the uterus. Generally we may readily restore the sunken fundus to its position, but it sinks back again to its former place when we let go of it. In some cases it cannot be restored to its normal position on account of peritoneal adhesions with the surrounding parts. Besides the flexion, there is almost always a slight anteversion or retroversion. If we cut the uterus out of the body, and hold it erect by the vaginal portion, the fundus sinks down, either anteriorly or posteriorly; if it be held horizontally, it not unfrequently holds its weight if the flexed portion be upward, but it bends together if we reverse it. The point of flexion is always near the internal os uteri. Here the flexion is sometimes slight, sometimes at right angles, or even at an acute angle. The os uteri is almost always moderately open, even in women who have had no children; this is a natural result of the tension on the anterior lip in retroflexion, on the posterior lip in anteflexion. The internal orifice, on the contrary, is contracted partly by the flexion itself, partly by the swelling of the mucous membrane. In older women, we occasionally find complete atresia of the internal os uteri. The contraction or closure of the internal os uteri causes more or less hydrometra. The disturbance of circulation at the point of flexion sufficiently explains the almost constant complication with catarrh of the uterus, ulcers of the os, and parenchymatous metritis.

Symptoms and Course.—The most constant symptoms of flexion depend on the impeded escape of the contents of the uterus. Hence the patients usually suffer severely from dysmenorrhœa as long as they continue to menstruate. Small clots of blood that has coagulated in the uterus are often mixed with the menstrual blood, which is evacuated with severe uterine colic. Uterine colic may also be caused, in the interval between the menses, by the impeded escape of the mucous and serous secretions, retained above the point of flexion. In many cases there are also the symptoms of uterine catarrh, as described in the first chapter, the fluor albus uterinus, decided loss of blood during menstruation, etc. Difficult and painful evacuation of the rectum, desire to urinate and pain while doing so, the signs of anæmia and bad nutritive condition, and finally the disturbances of innervation that have been so often mentioned, complete the description given by many women suffering from flexion of the uterus. But we must add that sometimes women with very decided flexions of the uterus never have any, or have but few, of these symptoms; or, if they do occur, they soon pass off without

the disappearance of the flexion. Sterility itself, although very frequent, does not constantly accompany flexions of the uterus. Hence it appears that it is not the flexions, but the other anomalies of the uterus, which complicate them, that cause the symptoms above described. These complications are so frequent that their absence is an exception. This is partly because the same injurious influences that induce the flexions also excite the different forms of metritis; partly because the disturbances of circulation at the point of flexion cause hyperæmia and exudation in the parenchyma, mucous membrane, and serous covering of the uterus. The course of flexions is very tedious. The disappearance of the condition is exceedingly rare, if it ever takes place. Flexion can only disappear completely if new parenchyma is formed in place of the flattened and atrophied uterine tissue. When, with advancing years, the periodical recurrence of physiological congestion of the uterus ceases, all the symptoms usually moderate; and when, in aged persons, the uterus and part of its blood-vessels become atrophied, the patient may feel pretty well.

On physical examination, the finger introduced through the vagina first feels a dislocation, anteriorly or posteriorly, of the portio vaginalis, caused by the coexistent anteversion or retroversion. We also generally find the os so patulous that the point of the finger may be readily passed into it, even in women who have had no children. From the vagina, either before or behind the vaginal portion, we may find the body and fundus of the uterus, forming a round, firm, movable tumor. Usually, also, we may reach the point of flexion. The fact that the opposite part of the vagina is empty prevents our mistaking a flexed uterus for one that is enlarged, or diseased in some other way. The introduction of the uterine sound aids the diagnosis; but when the uterus is flexed this operation is peculiarly difficult, and we again repeat that, in the hands of an unskilful physician, or even in those of a skilful one, the uterine sound is a dangerous instrument, which should be used as little as possible.

Treatment.—We have already said that total disappearance of a flexion must to a certain extent be considered as a physiological impossibility, as it is almost always accompanied by atrophy of the parenchyma of the uterus at the point of flexion. "Flexion instruments," sounds, redressers, and intra-uterine pessaries, which were for a time much used for flexions, do actually no good, but much harm. *Scanzoni* has stated plainly that in his large gynecological practice he has never cured a flexion, and that he considers the use of flexion instruments as of no use, and dangerous. Tonics also,

whether used locally or internally, promise no benefit. The advice that patients with anteflexion should hold their urine as long as possible, so that the distended bladder may raise up the fundus, and that patients with retroversion should retain the fæces for the same reason, is given from theoretical grounds, and has not proved correct. (*Virchow* says that anteflexion *results* from great distention of the bladder while the uterus is fixed.) Most gynecologists advise wearing a firm girdle around the pelvis and lower part of the abdomen, and many patients praise the results of this treatment. It is difficult to understand that, even in retroflexion, pressure on the lower part of the abdomen should relieve the patient. Perhaps their greater ease may be due to the compression of the uterus and its consequent bloodless state.

Little as we can do to remove the flexions, we may do much to relieve the sufferings of the patient if we treat the catarrh and parenchymatous inflammation of the uterus, which first call attention to the flexion, according to the rules laid down in previous chapters. Occasional abstractions of blood from the portio vaginalis are peculiarly beneficial, and are almost always effective in cases of flexion.

[To allay the distressing menstrual colic caused by retention of clots at the constricted point of flexure, most gynecologists resort to mechanical dilatation of the cervical canal. According to the experience of *Sims* and others, bloodless dilatation by means of bougies is less beneficial than, and fully as dangerous as, incision of the neck by the metrotome.]

CHAPTER VI.

CHANGES OF POSITION OF THE UTERUS.

The uterus, which is very movable, may be dislocated in any direction. The most important deviations in position are anteversion, retroversion, descent, and prolapse.

Anteversion most frequently occurs in women with strongly curved pelvis. The natural inclination forward of the uterus may be increased by the pressure from above of fluids in the peritoneal sac or of tumors, and anteversion may thus be caused. More frequently the uterus sinks forward from its own weight; hence almost always moderate degrees of anteversion accompany infarctions and new formations in the fundus uteri. Anteversion rarely becomes excessive, since each distention of the bladder restores the uterus to place, if some peculiar circumstance does not interfere with

the reposition. Corresponding to this, the annoyance caused by the disease is usually slight, and it is only when the uterus is otherwise diseased and enlarged, or when it swells up at the menstrual period, that compression of the pelvic organs and tension of Douglas's ligaments cause pain in the small of the back, pressure in the pelvis, difficult micturition, desire to go to stool, and pain during defecation. On vaginal examination, we find the portio vaginalis directed backward toward the hollow of the sacrum, and passing the finger forward we come without interruption upon the body and fundus, which lie against the anterior part of the vagina.

Retroversion depends on the same causes as anteversion. A continued pressure from before backward, tense adhesions on the posterior surface of the uterus or tumors in its posterior wall, cause a sinking of the fundus uteri into Douglas's space, and a prominence of the vaginal portion toward the symphysis pubis. The retroversion that occurs independently during the first months of pregnancy, and soon after confinement, is a very important disease; that occurring at other times is generally only a subordinate result of other diseases of the uterus or other pelvic organs. The symptoms of retroversion are analogous to those of anteversion, and they also depend on the pressure of the horizontally-displaced uterus on the pelvic organs, particularly on the rectum and bladder.

Descent and *prolapse* of the uterus depend chiefly on relaxation of the parts that maintain the uterus in position, particularly its ligaments, the pelvic fascia, and the vagina. If, during this relaxation, a stronger pressure from above downward act on the uterus, the latter is pressed down, inverts the vagina, and passes deeper into it, and the result is a descent. If part of the uterus protrude from the vulva, we speak of a prolapse; when the entire uterus lies outside of the vulva, it is called a prolapsus completus. Relaxation of all the parts that should maintain the uterus in position occurs most frequently in the puerperal state; and it is the abdominal pressure that most often presses the uterus downward. Poor women, who cannot take care of themselves after confinement, but are obliged to do hard work that causes abdominal pressure a few days subsequently, are peculiarly liable to descents and prolapses. As every descent of the uterus inverts the vagina, and each prolapse of the uterus induces prolapse of the vagina, so, on the other hand, prolapse of the vagina may give rise to descent or prolapse of the uterus. If the lower end of the vagina be prolapsed, as a result of rupture of the perinæum during delivery, or from some other cause, the upper end exercises traction on the uterus, which either results in elongation of the vagina or descent of the uterus. De-

scent or prolapse of the uterus only exceptionally occurs in women who have had no children. When they do occur, it is because the same conditions exist as after confinement, and particularly because the vagina is relaxed by blennorrhœa and venereal excesses, or the uterus is pressed downward by heavy tumors in the pelvis. If prolapse of the uterus take place suddenly, as occasionally happens from lifting heavy weights, from severe coughing, or strong bearing-down efforts, the stretching of the ligaments causes severe pain in the abdomen, and great general disturbance, fainting, nausea, etc. If the descent or prolapse develop slowly, there is usually but little annoyance at first; this consists chiefly in an undecided feeling of pressure downward, and in stretching pain in the lower part of the abdomen and small of the back. The deeper the uterus descends, the more annoying these symptoms become; they increase when the patient stands up, walks, coughs, etc; they diminish during rest, and in the horizontal position. There are also inconvenience and pain during urination and defecation, and there are constipation, colic, and other symptoms due to dislocation, pressure, and tension of the abdominal organs. If the uterus project from the vulva, it at first forms a round or oval tumor, as large as a walnut, which may be easily replaced; but soon the uterus covered by the vagina advances, the tumor rapidly increases in size, and is replaced with difficulty; it feels doughy, but on firm pressure we may distinguish a hard body more deeply seated. If, with the anterior wall of the vagina, the fundus and posterior wall of the bladder be drawn through the vulva (cystocele), on the anterior wall of the prolapse we may see a tense and sometimes fluctuating tumor, which swells and subsides again several times during the day, and into which, by exercising some skill, we may introduce a male catheter through the urethra. On introducing the finger into the rectum, we find it bulged forward. The os uteri gapes open, because the vaginal portion is averted; it is reddened and covered with glairy mucus. The prolapsed vagina is dry, parchment-like, thickened; the epithelium resembles epidermis; frequently it is excoriated by the friction of the clothes and the irritation of the urine that trickles over it, and not unfrequently deep ulcers form in it.

The treatment of dislocations of the uterus is purely surgical; and it would lead us too far to speak even of the choice of certain pessaries, and of the rules for their introduction and employment.

CHAPTER VII.

FIBROID TUMORS OF THE UTERUS—FIBROMYOMATA, FIBROIDS, DERMOIDS.

[Pathology and Anatomical Appearances.—These growths are to be regarded as partial hyperplasias of the uterine parenchyma, and consist of masses of smooth muscular fibres intermingled in varying proportion with connective tissue. In their original form they always present rounded, sharply-defined tumors. They are non-malignant in their character, and are far more common than any other form of uterine growth. They rarely appear before puberty, and their liability to develop increases up to a certain point with the age, so that they are most commonly found in persons from thirty to fifty years old, while beyond this age they are comparatively rare. They are met with both in the married and unmarried, and are often associated with menstrual disorder and sterility, which, however, must be looked upon as consequences rather than as causes of the disease. Nothing certain is known regarding their cause. Fibromyomata vary greatly in size. They may be no larger than a pea, or may form a tumor as large as the head, and may weigh several pounds; but this is somewhat rare. They are sometimes solitary and sometimes multiple, and in the latter case they are usually small.

The constitution of a fibromyoma varies. Sometimes the fibrous element preponderates, giving it more the character of a pure fibroma; sometimes (but rarely) the growth consists almost purely of unstriped muscular fibres, thus approaching the true myoma in nature. Upon section, their structure is found to consist of more or less regular concentric layers of wavy connective tissue, between which the finer bundles of muscular fibres are arranged. The more the firm, tendinous, fibrous tissue preponderates, so much the harder is the tumor and the paler its cut surface. When the muscular element prevails, the section presents more of the grayish-red coloring of the normal uterine tissue. As a rule, fibromyomata are but sparingly vascular, and very seldom contain large blood-vessels. There is, however, one form in which these tumors consist, either partially or totally, of enormously dilated blood-vessels. Sinuses filled with blood may even form, giving to the tumor a likeness to the erectile tissue of the corpus cavernosum, and also its capacity to swell and shrink; so that sudden changes of volume in such a tumor can be observed (*Virchow's* telangiectatic or cavernous myoma).

Fibromyomata often exist in a conglomerated form. The tumor is then irregular and knobby in contour, instead of rounded. This shape is sometimes attributable to the blending of several independent tumors; but more commonly the self-same morbid process which causes the original growths in their simpler form again repeats itself in the substance of the new growth. Each vessel, with its appertaining connective tissue and muscular fibres, proliferates, and begets, as it were, a second generation of nodules within the older ones. Although the fibromyoma springs from the uterine parenchyma, yet at a later stage it no longer remains closely connected with it, but lies within the uterine substance, though quite isolated from it by a layer of loose areolar tissue, so that it can very easily be enucleated. Sometimes, however, old fibroids of large size maintain their connection with the womb by a pedicle, or even by a broad fibromuscular band. Such an attachment usually contains large blood-vessels. The womb is usually hypertrophied, but in advanced age it is atrophied. Now and then it is elongated and thinned, and it is often out of place.

Fibromyomata rarely spring from the cervix, but rather from the fundus or body. Their classification according to their more superficial or deeper seat in the uterine wall is of great practical importance. Thus we distinguish (1) the *subserous*, (2) the *submucous*, and (3) the *interstitial* fibroids, although many tumors present intermediate forms. In the subserous fibroma, the tumor, which usually has originated in the outer layers of the womb, tends toward the abdominal cavity, into which it projects in strong relief. Its base, at first wide, may by degrees grow narrower, and ultimately even present a mere stalk of the peritonæum. Total detachment, indeed, has been observed; but in such cases the growth has soon formed new adhesions, either in the vicinity of its point of origin or in Douglas's cul-de-sac. Subserous fibroids are often multiple, and usually are small; but if their vascular connection with the uterine parenchyma, by which they are nourished, should happen to be kept up, they may attain a great size, and rise into the abdomen. By these large subserous tumors the womb is misplaced, distorted, and stretched; but it is almost never hypertrophied. When springing from the side of the womb, they may penetrate between the two folds of the broad ligament, forcing them asunder so as eventually to lie quite outside of the peritonæum. The submucous fibroid, on the contrary, tends to grow inward, toward the cavity of the womb, into which it juts like a globe, distending it, and displacing it to the side opposite to that whence it springs. Its surface is covered either by mucous membrane alone, or by a thin

layer of parenchyma. The womb itself is usually hypertrophied as if pregnant, and the portio vaginalis is soon obliterated. Practically it is of great importance to distinguish between submucous growths having a broad attachment to the womb, and those which project into it so completely as to be held by a mere pedicle. The latter, the so-called fibrous polypi, are a subject for surgical treatment. The interstitial, intraparietal, or intramural tumors tend less to project from the uterine wall; but, where they become large, they cause a bulging of both inner and outer wall, thus taking an intermediate position between the two extraparietal forms of fibromyoma.

The same variation as to seat holds good with regard to the far rarer fibromyomata of the neck and vaginal portions of the womb. Submucous fibromata of the cervix usually appear as fibrous polypi. These must not be confounded with the more common mucous polypus, which is merely a product of catarrhal relaxation of the mucous membrane with cystic enlargement of the follicles (ovula Nabothi). The latter are easily distinguishable from the fibromyomata by their softness and smallness, rarely exceeding a hazel-nut in size.

Uterine fibromyomata may undergo many further transformations. The commonest one is an accumulation of liquid in the lymph-spaces, which dilates them, causing an œdematous softening of the tumor, and sometimes a cystic metamorphosis. In the latter case we find either numerous small cysts, or else a large cavity whose ragged sides and trabeculæ announce it to be the product of the blending of several smaller cysts. Heteroplastic action may also arise, such as myxomatous or colloid degeneration, sarcoma, or cystosarcoma. True cancer probably does not occur, except as a secondary disease, by metastasis or by propagation in continuity from a cancer of the uterus proper.

Symptoms and Course.—The symptoms of a fibromyoma vary greatly, according to the size and seat of the tumor. Small subserous growths, or those which are completely extraparietal, and which, being merely attached by a thin stem, have ceased to grow, often give no symptoms at all. Sometimes a small growth flexes or upsets the womb by its weight, either backward or forward, according as its seat is in the front or back wall of the organ. A large extraparietal tumor, on the other hand, displaces the womb in the direction away from its seat, or sometimes draws the womb up with it into the abdomen. The pressure exerted by a dislocated womb or by a voluminous tumor upon adjacent parts may cause various evils—backache, incontinence or retention of urine, obstruction of the rectum, swelling of the feet, neuralgia, or even paresis of a leg. The irritation often causes local peritonitis, with consecutive incap-

sulated exudation, or with adhesions; and these latter may by and by hinder the tumor from lifting itself as it grows out of the pelvis, so that the pressure upon the other pelvic organs may give rise to the symptoms of incarceration. All these pressure-symptoms usually grow worse during the menstrual period.

The submucous fibromyomata also soon create serious distress. In this form the distended and hypertrophied womb itself forms the tumor, and when large it naturally produces the same pressure-symptoms as those caused by subserous tumors. But the growth, being enclosed in muscular masses, excites the latter to strong contraction. The tumor, projecting into the womb, stretches and irritates its mucous lining, thus inducing leucorrhœa and hæmorrhages of great frequence and severity. These metrorrhagias occur even when the tumor itself is very poor in blood-vessels, and proceed entirely from the mechanically overladen vessels of the mucous membrane. At first the bleedings seem to attend the menstrual periods, which are then excessive and followed by a watery mucous discharge. By and by the hæmorrhages recur irregularly, and with ever-increasing frequence and severity, so that the patient's life may be jeoparded by sheer loss of blood. Great suffering during menstruation also arises in this form of fibroma. There is pain, which is sometimes continuous, and sometimes an intermittent uterine colic, like labor-pain, due to the obstruction by the tumor to the menstrual flow. If the catamenial function ceases permanently—and this is apt to take place quite late in life in these cases—this distress is materially abated or ceases entirely. Interstitial tumors occupy a middle position both as regards seat and symptoms. Being well surrounded by uterine tissue, and being under more favorable conditions for growth than the subserous and submucous tumors generally are, their development is often quite rapid, and they sometimes attain colossal dimensions.

The existence of a fibromyoma cannot be made out without manual examination, and even then it is sometimes very difficult. The subserous tumor is felt as a hard ball-like or nodular lump, which, if it spring from the fundus, can be felt more easily through the abdominal wall, and if from the lower part of the womb, is best found through the fornix vaginæ. The examination should aim to prove that the tumor really proceeds from the womb, and does not merely lie next to it. Sometimes an ovarian cyst or an old parametritic or perimetritic exudation adheres so closely to the uterus that nothing will shed light upon the case but a due weighing of other facts, such as the consistence of the mass, which, if a fibroma, should be hard, and never is soft unless it be in a state of cystic degeneration,

when it is more like an ovarian cyst in consistence ; or else the history of the case would show a preëxisting pelvic inflammation, had any occurred. Subserous fibroids attached only by a thin fibrous stem are often thought not to belong to the womb, and, although too hard to be mistaken for ovarian cysts, may be difficult to distinguish from a solid ovarian growth. The examination should further determine that the object felt is not the womb itself, displaced and enlarged. An anteflexed or retroflexed womb may simulate a tumor lying in the uterine wall ; but conjoined manipulation will demonstrate that beside the tumor in question a second one exists, which, in situation, form, and consistence, corresponds to the womb ; and, if necessary, the introduction of a sound will show by its direction that it does not enter the tumor, but takes an entirely normal course. Sometimes by means of the sound we can tell that the uterus, although distorted and displaced, by no means corresponds in length with the volume of the tumor.

Submucous fibroids lie hidden within the womb, and distend it ; hence they may be confounded with other conditions which cause uterine enlargement—above all, with pregnancy, uterine infarction, and hæmometra. Unless the history of the case and other circumstances distinctly preclude the supposition, the tumor may be mistaken for a gravid womb, because the same general disorders which characterize pregnancy, as well as the changes in the breasts which then arise, may likewise be met with now. The condition of the vaginal portion is of great value in the diagnosis. In pregnancy it is remarkably soft and flabby. In a case of fibroma the cervix rapidly disappears, and the os uteri will gape so that the tumor within it can easily be found. The uterine *souffle* so regularly heard in pregnancy is, nevertheless, not a sure sign of that condition ; for it also is heard in cases of fibroma, and in rare instances where there is an ovarian growth. In chronic metritis the tenderness of the womb to pressure stands in contrast to the insensibility of a fibroma. Moreover, in infarction the vaginal portion is thickened and not obliterated, and the sound enters easily ; whereas a fibroma opposes an obstacle to its passage, not always readily overcome.

After excluding pregnancy, the diagnosis may be sometimes facilitated by the introduction of a sponge-tent into the cervix. The canal being thus dilated, it becomes practicable to examine precisely the tumor with the finger and sound, and particularly to determine whether the tumor is attached by a broad base or by a slender pedicle—a matter of the utmost importance as regards therapeutics and prognosis. Interstitial tumors bulge less distinctly

into the uterine cavity, and the enlargement of the organ is not so general and uniform. Very large interstitial fibromata, which rise into the abdomen, may be mistaken for other abdominal growths; not for ovarian cysts, however—which, though hard in places, also as a rule present elastic, fluctuating portions to the touch—but rather for solid ovarian tumors and for subserous fibromata. But in these two cases the womb itself does not form part of the tumor, but is merely displaced by it; and we must strive to ascertain if the two bodies can be made to move independently of one another. If so movable, the growth is probably ovarian; if attached, it is most likely a subserous fibroid; but no absolute certainty of this can be attained, because an ovarian growth may be bound fast to the womb, and a subserous fibroid with a long pedicle may float freely. When the uterine sound passes deeply into a tumor, or when its tip can be felt through the walls of the growth, we may infer that the mass is not merely attached to the uterus, but is the uterus itself. An expert with the sound can also determine that the enlargement of the organ is not uniform, but chiefly affects one or other wall. This decidedly points toward the presence of an interstitial fibroid.

Fibromyomata of the cervix, which are more rare, are easy of diagnosis. They present either rounded, hard, circumscribed lumps, which distend one of the lips of the os, or else appear as fibrous polypi protruding from the cervix.

Prognosis.—The prognosis of uterine fibromyomata has its good and its bad points of view. The fact is well attested that such tumors have now and then shrunk away and disappeared, but such good fortune must never be counted on; the more so, because these rare cures seem never to be due to treatment, but rather to favorable accidental conditions—parturition, the climacteric, or the like.

The affection is generally to be regarded as incurable; yet the prognosis is so far favorable that life is seldom immediately imperilled by it. Sometimes, indeed, the envelope of the tumor suppurates, causing peritonitis or pyæmia; and the general nutrition has been known to suffer seriously through the pressure of an enormous fibrous tumor upon adjacent abdominal organs. The tumor sometimes ceases to grow so early as never to cause any symptoms; while others by their development render life a burden, until the attainment of the climacteric brings about an arrest of development, or until a sort of imperfect retrogression takes place by calcification of the tumor. Spontaneous expulsion of the growth through the uterine canal has been observed in a series of cases. A few times it has been known to be expelled into the abdomen or into the bladder, and even to be discharged through the abdominal walls. Such

an event is either the result of sloughing and suppuration of the capsule, with subsequent bursting of the resulting abscess, in which case the danger to life is very great, or else the extrusion comes to pass by the less dangerous method of spontaneous enucleation. A tumor juts from the os ; at its apex an opening forms by sloughing of the mucous membrane, and through this opening, which enlarges more and more, the tumor is gradually pushed by the uterine contractions. It sometimes happens that pregnancy occurs notwithstanding the presence of a fibromyoma. Childbed in such a case is beset with peculiar dangers.

Treatment.—Very little reliance is to be placed upon medical treatment of a fibromyoma with a view to its dispersion, arrest, or atrophy. Iodide of potassium, bromide of potassium (first proposed by *Simpson*), chloride of calcium (*Rigby*), arsenic and phosphorus (*Guéniot*), seem to effect very little ; while the brine-baths, particularly those containing bromine and iodine (Kreuznach, Münster am Stein, and Adelheidsquelle in Bavaria), seem sometimes to bring about a certain reduction in bulk of the tumor. *Hildebrand* claims that by means of subcutaneous injections of ergotine, employed originally to control the bleeding, not only the hæmorrhage, but also the tumor, was made to disappear ; but, in spite of other corroborative testimony, the value of this treatment is not yet established. Some fibromyomata are curable by surgical treatment ; and by following nature's suggestion of spontaneous enucleation, not only submucous tumors, but even interstitial ones, have been removed through the vagina by a surgical operation, though at considerable risk to life. As a last resort, indeed, to save life, when the rapid growth of the tumor was obviously about to kill the patient, extirpation of the tumor, either with or without the womb, has been successfully accomplished.

The surgical treatment of pedunculated submucous tumors is more satisfactory ; and we not seldom meet women, who have been brought almost to death's door by persistent hæmorrhage from fibrous polypi, restored in a few months after their removal to a state of blooming health.

As a rule, however, we must renounce the expectation of actually curing a fibromyoma, and content ourselves with enjoining a quiet mode of life, with little physical exertion or excitement, forbidding frequent sexual intercourse, and with practicing small local blood-letting upon the vaginal portion, with regulation of diet ; also with the recommendation of a treatment during the summer-time by baths calculated to retard the growth of the tumor. Regarding the treatment of the bleeding, we refer to the article upon menorrhagia.]

[CHAPTER VIII.

CANCER OF THE WOMB.

Etiology and Anatomical Appearances.—Primary cancer appears more frequently in the womb than in any other organ of woman, not excepting the breast. Secondary cancer of the womb, on the other hand, is rare. Nothing certain is known as to the cause of this miserable disease, and its assumed hereditary transmissibility is not proved. It is met with chiefly among married persons, especially among multipara, and with increasing frequency from the thirtieth year up to the climacteric period, which age furnishes the most victims. Beyond it cancer is common enough still, and does not become a rarity until the seventieth year.

The point of origin of primary cancer is almost always in the neck of the womb, very rarely in its body. According to *Waldeyer*, all forms of cancer spring from the true primordial epithelium of the organism, and hence are to be regarded generally as epithelial growths; and even the smooth and papillated cancroids formerly distinguished as carcinomata are to be included in this category. The development of cancer of the cervix is as follows: The superficial epithelium of the mucous membrane proliferates, throwing offshoots into the connective and muscular tissue beneath it. These sprouts, ramifying in all directions, everywhere form new nests of cancer-cells, and encroach more and more upon the normal tissues, which finally break down and ulcerate through pressure and arrest of circulation. The cancerous infiltration meanwhile continues to invade new tissues around it, which in their turn ulcerate. This is often accompanied by considerable development of the papillæ of the mucous membrane, and this is in the beginning liable to be confounded with the non-malignant papillary outgrowth due to catarrh. Indeed, it has been assumed that papillomata, originally simple, might change into cancer by consecutive ingrowth of their epithelium into their basement-tissue. At a later period the malignant papilloma is distinguishable from the simple form by the rapidity and luxuriance of its growth, and by its early ulceration. At other times, instead of papillæ, there develops a smooth, knotty group of swellings in one or other lip, or perhaps throughout the whole circumference, of the os. These nodules, unless they prematurely break down by ulceration, may form a tumor which fills the upper part of the vagina. But there are cancers which do not begin at the surface, but originate deep in the interior of the neck, and develop as circum-

scribed knots pushing toward the surface. Even this form of cancer, however, must derive its origin from preëxisting epithelium, probably from isolated cervical glands, and from abnormally distributed vestiges of the primordial epithelial germ. Sometimes the disease takes the form of a firm scirrhus, but more commonly it assumes that of a soft medullary cancer, with a predominance of epithelium and with scanty connective tissue. The infiltration and ulceration travel upward to the internal os, and sometimes even invade the body of the womb. Thus not the neck alone, but a large part of the body of the organ, may disappear. An extension of the disease to the vagina imparts to the latter a hard, puffed-up, brawny quality, so that the finger can no longer recognize the os uteri. Still later, the cancerous infiltration of the ureters often causes obstruction of the urine and hydronephrosis (even to the extent of fatal uræmia), and, by invasion of the bladder and rectum, with consequent destruction of the septa separating these cavities, large fistulæ are formed; and it has even happened that all three cavities have been blended into one horrible cloaca. The tubes, ovaries, and other pelvic organs, and the muscles, areolar tissue, and even the bones, are finally attacked, either by contiguity or by metastasis; so that at last the womb is, as it were, walled up by cancerous infiltration and peritoneal adhesions. More distant regions finally are invaded—the inguinal and retroperitoneal glands, the peritonæum, omentum, mesentery, intestine, liver, and perhaps even the breast.

Symptoms and Course.—In the earliest stages of uterine cancer there is slight pain in the back and abdomen, the menstrual flow is augmented, and there is leucorrhœa, which is not at first profuse, except in a case of malignant papilloma. So little is presented to distinguish it from the more unimportant disorders of the sexual organs, that in the beginning the malady is rarely recognized. It would seem, indeed, that that form of it which commences by deep-seated nodules presents no symptoms whatever so long as the mucous membrane remains intact. In many cases no serious symptoms arise until the cancerous mass begins to ulcerate and break down; so that the period for operative interference has already passed ere the nature of the malady has been recognized. Indeed, even in the earlier stages, while the mucous membrane remains sound, a local examination will not always reveal the existence of cancer, and the most experienced gynecologists admit that the distinction between a cervix enlarged by cancerous infiltration and a simple hypertrophy of the neck may at first be impossible. *Schroeder* describes a condition of the mucous membrane as characteristic of cancer even before the period of ulceration. The membrane is

tightly adherent to the tissues underlying it. As *Waldeyer* has it, the mucous membrane seems to be tacked down by the ingrowing epithelial sprouts, which penetrate the tissues beneath them like so many brads. After it has attacked the vagina and surrounding connective tissue, the recognition of cancer is easy.

When the period of ulceration sets in, a series of grave and often very distressing symptoms arise. The leucorrhœa becomes more profuse, and somewhat sanguinolent (like meat-washings), and finally, by admixture of gangrenous particles, acquires an ichorous character, and assumes a darker dirty-brown or greenish-black color, and emits so foul and putrid an odor as to disgust not only the neighbors but the patient herself. Hæmorrhage is rarely absent. In the beginning there is merely an augmentation of the menstrual flow, but the bleeding afterward becomes irregular, and is often the first symptom which alarms the patient. Elderly women, however, are prone to ascribe such bleedings to the irregularities common at the menopause, and hence give themselves little concern over it. Only in cases of hard cancer is hæmorrhage ever absent. It is most profuse in the papillary form. Notwithstanding its frequence and abundance, which often reduces the patient to the lowest stage of anæmia, a fatal hæmorrhage is rare.

The abdominal and lumbar pains are sometimes insignificant throughout the disease ; while at other times the patient suffers indescribable agony, especially during the night. This violent pain does not depend upon the ulceration, but upon extension of the cancerous growth into the connective tissue and other parts of the body, and appears to be due to pressure of the indurated mass upon the nerve-twigs which it encloses. Besides this, there may be sharp pain from pressure set up by a secondary peritonitis, arising from the irritation of the cancerous nodules. Finally, the pain may assume a paroxysmal form—a uterine colic—in consequence of an obstructed outflow of blood and secretion from the womb. Incessant and painful urinary irritability indicates invasion of the bladder, while a suppression of the urine makes it probable that the ureters are occluded by the growth. Obstinate constipation, followed by tenesmus, hæmorrhoids, and diarrhœa, indicates an implication of the rectum.

Prior to the stage of ulceration the general health of the patient is fairly comfortable ; afterward she grows pale, anæmic, œdematous, and emaciated, and the countenance often presents that dirty-yellow coloring so commonly seen in cancer, although not a trustworthy sign of it. The more frequent the bleedings, the more profuse the discharge, and the more constant and severe the pain,

so much the sooner does the general health of the patient give way. There is also almost always a derangement of the stomach, with loss of appetite and loathing of food, and a tendency to nausea which contributes to break down the strength; and it is often hard to determine whether the vomiting is due to a sense of disgust, or arises from gastric catarrh or gastric irritation reflected from the genital organs, or from the effect of uræmic poisoning.

Ulceration usually first attacks the inner surface of the lips of the os. After this has once begun and loss of substance has taken place, the diagnosis by digital and ocular examination is no longer difficult. One source of error might be the destruction of the vaginal portion of the cervix by that very rare form of ulcer (*ulcus phagedænicum corrodens*) first described by *Clarke*, and identified with certainty in a few instances only by *Klebs* and *Foerster*, and the cause of which is still unknown. In a doubtful case, nothing but the microscopic examination of an extirpated portion of the cervix can determine whether the base of the ulcer contains traces of carcinoma or not. But, after all, the prognosis of a corroding ulcer is scarcely better than that of cancer; for our experience has shown that neither by the strongest caustics, nor even by the hot iron itself, can the ravages of the latter be checked and the ulcer healed.

As we do not generally know how long the latent stage of cancer has existed, its duration can rarely be stated with certainty. Dating from the time of first discovery, *Gusserow* estimates that most patients succumb in about a year or a year and a half. Yet there are examples of a rapid course of only a few months, and others again of several years' duration. Death usually takes place from exhaustion, or else the patient dies of complications. These are acute or chronic uræmia, gradual or perforative peritonitis, miliary carcinosis of the peritonæum, septicæmia, dysentery, diphtheritic inflammation of the vagina or bladder, pneumonia, or (what is more rare) embolism of the pulmonary artery, embolic gangrene of the lungs, fatty degeneration of the heart, pyelonephritis, or pylephlebitis as a consequence of putrid thrombosis of the portal vein.

Treatment.—The treatment of cancer of the womb is essentially surgical, and an operation, although it very rarely cures the patient, yet affords the only chance of effecting even a temporary improvement. Unfortunately, the malady is seldom brought under treatment until the vagina has become implicated, or until so much of the cervix is involved that an amputation can no longer be made through healthy tissue. Yet even then the operation is justified as affording a last chance for prolonging life. This is best done by

removing all parts which seem affected by knife or scissors, and by scooping away the less accessible portions by means of Simon's sharp spoon, and then by cauterizing the wound with the hot iron, or by means of the alcoholic solution of bromine first recommended by *Routh*, and approved by *Schroeder* and *Henneberg*. Otherwise the escape of cancerous offshoots which often exist in parts seemingly healthy, or of remnants of the ulcer which often extend up to the inner os, may render the operation abortive. The relief of pain is the most important duty in treatment of the symptoms. Hypodermic injections of morphine and opiate enemata are often quite indispensable. When the flow is profuse, copious astringent vaginal injections are demanded ; and for the foul odor we must employ disinfectants, such as carbolic acid, permanganate of potash, and chloride of lime. The hæmorrhage is to be combated by the use of ice, or of cold or very hot injections, or of dilute liquor ferri sesquichloridi ; or else we must tampon with sponges, or plugs of cotton charged with strong liquor ferri chloridi. For the insomnia which is so common, chloral furnishes an invaluable remedy.]

CHAPTER IX.

ANOMALIES OF MENSTRUATION.

[It has generally been assumed that the menstrual flux is intimately connected with the ripening and bursting of a Graafian vesicle, and with the escape therefrom of one or more eggs ; but this view of the subject has latterly been contested, and the complete independence of ovulation and menstruation has been asserted (*Beigel*). According to our present information upon the subject of ovulation, the uterine mucous membrane at puberty undergoes certain periodical metamorphoses, the nature of which has been ascertained by the researches of *Kundrat* and *Engelmann*. They find that at the menstrual period the uterine mucous lining swells up and develops ; its glands elongate and dilate, and a proliferation of the round cells of its basement-tissues takes place. This process is followed by a retrogressive one, based upon a fatty degeneration of the upper layers of the mucous membrane, so that the bleeding is caused, not, as is generally supposed, by a menstrual fluxion and hyperæmia, but by destruction of the mucous membrane through fatty degeneration. The purpose of the increased development of the mucous membrane is that the egg may be received into it, and only at this time of so-called *nidation* is the growth of a fructifying egg

possible; while in the remaining period, that of so-called *denidation*, during the menstrual flow and during the exfoliation of the mucous membrane, any egg which may reach the uterus will perish.

Hence it would at first seem as though conception could only take place after a coitus immediately preceding menstruation, and never from coitus following it; but we should bear in mind that the ripened egg which leaves the ovary at the menstrual epoch requires a certain period of time, not precisely known, for its passage through the tube, at the end of which period the state of denidation may have passed over. According to the newest views, those of *John Williams*, based upon examination of twelve wombs, the regeneration of the fatty mucous lining begins immediately after cessation of the bleeding, and progresses steadily until the next retrogressive period commences. Thus the womb would seem never to be at rest, but to be either in a state of evolution lasting about three weeks, or of involution lasting about one week. Only at the period of involution would implantation and fixing of the ovum (as far as the womb is concerned) seem to be impossible, while impregnation might take place at any part of the period of evolution. *Bischoff*, however, doubts if an egg be capable of fructification during the latter part of the period of evolution. More probably the egg must receive an impulse toward further development from the seminal fluid while still in the tubes, and only in the improbable event of an egg's requiring three weeks to traverse the tube would there be no period of intermenstrual unfruitfulness.]

Anomalies of menstruation are not independent diseases, but are symptoms of affections of the sexual organs, or of other diseases impairing the general health. Hence the discussion of amenorrhœa, dysmenorrhœa, menorrhagia, etc., does not properly belong to a text-book of special pathology and therapeutics, but to a work on semeiology and diagnosis. Following the example of most authors, for practical reasons, we will give a short account of the most important menstrual disturbances.

Too early menstruation—*menstruatio præcox*—is not frequent, if by this term we mean only those cases of hæmorrhage from the female genitals before puberty that are accompanied by the expulsion of a ripe ovum. As we have no certain means of finding out whether this complication exists or not, we must note whether the hæmorrhage recurs at regular intervals; whether it is accompanied by disturbance of the general health, by pains in the back, and other symptoms which almost always accompany menstruation proper. Hæmorrhage occurring once, or at irregular intervals in the course of acute diseases, particularly of acute infectious dis-

eases, as well as in chronic dyscrasia and venous congestions, has nothing to do with menstruation. If, in our climate, menstruation begins between the twelfth and fourteenth years, instead of the fourteenth and sixteenth, it is only a morbid symptom if the girl be undeveloped. Many girls at this age, who still go to school and wear short clothes, have full breasts and hair on the pubes. We may say that they have developed too early, but not that they have any anomaly of menstruation; in them the absence of the menses would be pathological. But, besides these cases, we not unfrequently find apparently undeveloped girls, aged eleven or twelve years, with regularly-recurring hæmorrhages from the genitals, and such characteristic symptoms of congestion in the pelvis that we cannot doubt there is a case of early ovulation—a true menstruatio præcox. Experience shows that almost all such girls subsequently suffer from obstinate chlorosis. Cases where menstruation has been observed in small children are only partially reliable.

It is very rare indeed for menstruation to cease several years too late. Among us, women usually menstruate till forty-five or forty-eight years old. If menstruation has begun early, it usually ceases somewhat sooner; if the reverse, it continues a few years longer. *Scanzoni* has seen only one case, in his practice, where an unmistakable menstrual hæmorrhage continued to the age of fifty-two years. Even very old women are inclined to regard all hæmorrhage from their genitals as menstrual.

Of course, we can only speak of *amenorrhœa* when the menses are absent in a woman who has attained the age of puberty, and has not passed the climacteric, and who is not pregnant or nursing. [Above all, be it observed that the absence of menstrual flow does not warrant the inference that ovulation has ceased. We know, indeed, that conception may take place during amenorrhœa. On the other hand, the cessation of ovulation after destruction of the ovaries is not always followed by absence of the menses. In exceptional cases menstrual bleeding has been observed to continue for some time after the sexual function has been destroyed.]

Tardy menstruation is one form of amenorrhœa; too early cessation is another form. If a girl sixteen or eighteen years old be no more developed than one of ten or twelve years, she can no more be said to have retarded menses than a fully-developed girl of twelve or thirteen who menstruates can be said to have menstruatio præcox. Except in cases of tardy menstruation or too early cessation of the menses, amenorrhœa more frequently depends on constitutional disease than on local affections of the genitals. It is chiefly chlorosis, scrofula, and tuberculosis that retard the occur-

rence of menstruation, or cause its arrest. It is not always easy to determine, in these cases, whether the ovum does not ripen, or whether only the hæmorrhage that usually accompanies the expulsion of the ovum is absent. If, at intervals of four weeks, we notice more or less decided molimina, accompanied by swelling of the breasts and increased discharge of mucus from the genitals, it would seem as if only the hæmorrhage were wanting; in the reverse cases, we may suppose that ovula do not ripen. Among the diseases of the sexual organs, degeneration of the ovaries rarely causes amenorrhœa, and only does so when both ovaries are at the same time the seat of organic disease. Among the various diseases of the uterus, chronic catarrh, and especially chronic infarction, in which the blood-vessels are compressed by the shrinking connective tissue, most frequently induce this disease. Amenorrhœa occasionally occurs in strong, healthy girls, in whom the genitals have not developed so rapidly as the rest of the body. Lastly, *Scanzoni* concludes, from the cases where the menses (which had previously been normal) ceased on the occurrence of paraplegia, that amenorrhœa may result from abnormal innervation.

The sudden arrest of the menstrual flow—*suppressio mensium*—is most frequently the symptom of acute metritis. It depends on the same injurious influences that we mentioned in the etiology of that disease, and is accompanied by the same symptoms. More rarely the menses cease suddenly, if the amount of blood in the uterine vessels be lessened by a diminution of the entire amount of blood in the body by venesection, or by excessive fluxion to some other organ.

[Sometimes, at the period when the menstrual flow should appear, instead of it a bleeding occurs from some other region, from a hæmorrhoid, from a wound or ulcer, from the nose, the bronchi, or the stomach. Such bleeding, it would seem, might properly be called "vicarious" so long as it was supposed that the menstrual flux had the effect of evacuating effete matter from the system. It might then have been inferred that refuse which was not voided by legitimate methods might be advantageously got rid of by unusual processes. Nowadays, however, with our present conceptions of the nature of menstruation, it is difficult to find a reason for a vicarious menstrual hæmorrhage. When we remember that the amount of the flow in all is only from about eight ounces to (at the utmost) about thirty ounces, the theory that the omission of an habitual menstruation can give rise to a general plethora must seem very rash. Most probably vicarious hæmorrhages stand in close relation to a very active general nervous and circulatory excite-

ment, accompanying ovulation, under the influence of which bleedings may take place at points predisposed to it, both when the menses are absent and when they are present.

The direct treatment of amenorrhœa by emmenagogues has undergone great limitation of late years. Many of the ancient physicians (mistaking cause and effect) were disposed to ascribe divers disorders to amenorrhœa as a cause, and to consider the reëstablishment of the menstrual flow a matter of great importance; whereas, in many a broken-down subject, it is rather to be wished that she may not be still further prostrated by loss of blood. Sometimes, however, in exhausted, bloodless patients, the growth of the Graafian vesicle is arrested, and the periodical ovulation with its uterine hyperæmia does not take place. Such amenorrhœas, arising from defective nutrition and anæmia, can only be successfully treated by allaying the main disease by tonics, iron, strengthening diet, fresh air, and the like. All the different emmenagogues possess in common the property of inducing hyperæmia of the pelvic organs, and especially of augmenting a congestion which already exists, and thus of producing a hæmorrhage. This object is most certainly obtained at the time when the menses should naturally return. This period is not, however, invariably one of twenty-eight days; for it may vary by several days in the same woman. Accepting the results of recent researches, that menstrual flow does not proceed from engorgement alone, but is also the effect of a fatty degeneration and exfoliation of the uterine mucous membrane, it becomes impossible to admit that the flux can be induced by emmenagogues at any other time than that of nidation.

Artificial induction of the menstrual flow may be called for when certain disturbances (pain in the back, local congestion, and the like) which normally accompany menstruation indicate a congestion of the pelvic organs, and that the hæmorrhage which should promptly relieve them has failed to arise. Measures calculated still further to augment the hyperæmia of the womb may then be resorted to to bring on the bleeding. These are irritating foot-baths, warm sitz- or full-baths, dry cups or mustard-plasters to the thighs, a hot or even a cold douche to the sacrum, clysters of aloes, or direct irritation of the womb by irritating vaginal douches. More rarely, painting the os with iodine, passage of a sound, or electricity may be tried; but we should be slow to resort to the internal exhibition of aloes or of savin, on account of their undesirable secondary effects. We must bear in mind, too, that, if we fail to induce the flow by these means, we may nevertheless so augment the uterine congestion as to aggravate the existing distress. Hence the use of

emmenagogues is only proper when the amenorrhœa is due solely to feebleness of the uterine congestion. In other cases the congestion is to be relieved by local depletion, leeches, or scarification of the vaginal portion, or in virgins by cups applied to the inner side of the thighs.]

Menorrhagia, or too copious menstrual hæmorrhage, has been already mentioned as a symptom of various structural changes and of tumors in the uterus. But it also occurs without perceptible disturbance of nutrition, where the escape of blood from the uterus is hindered in heart and lung diseases, etc., as well as in fluxions induced by irritation of the uterus from sexual excess, perhaps also by sensual excitement. In very rare cases menorrhagia depends on a hæmorrhagic diathesis, being a symptom of scurvy, purpura hæmorrhagica, acute infectious diseases, such as hæmorrhagic smallpox or measles, or of typhus, etc. Where it is due to obstructed efflux or increased afflux of blood to the uterus, it is usually preceded by symptoms similar to, but more marked than, those with which normal menstruation usually begins. Part of the blood passes off in a fluid state, part coagulates in the vagina, forming irregular clots ; rarely it coagulates in the uterus, and there forms so-called fibrinous polypi, such as frequently appear after abortions. Plethoric patients often bear very decided loss of blood without injury ; anæmic patients have the symptoms of increased anæmia. The treatment of menorrhagia requires great attention to the original disease. The occasional application of leeches to the cervix is often of surprising benefit in those cases dependent on chronic inflammation of the uterus. As soon as the loss of blood is decided and threatens to impair the strength, it is important to prevent all bodily exertion and mental excitement, and to keep the patient in a horizontal position during menstruation. At the same time we forbid all stimulating food and drink, and order mineral or vegetable acids. It will only rarely be necessary to employ cold water or ice-compresses, or to have recourse to styptics. In some cases, however, the bleeding threatens life, and then it is necessary to act energetically, and even to inject hot-water solutions of chloride of iron, etc., into the uterus.

[The term *dysmenorrhœa* is applied to the conditions under which, instead of the trifling disorders which are almost normal, grave and distressing symptoms arise, especially violent pain in the back, belly, groins, and thighs, and sometimes sympathetic derangement of remote organs, such as cramp in the stomach, vomiting, diarrhœa, numbness, headache, dizziness, and convulsions. The causes of dysmenorrhœa are numerous. It has been assumed that

a menstrual congestion of extraordinary activity might give rise to pain in the womb and ovaries, by irritating the nerves of those organs (congestive dysmenorrhœa). In other cases we are reduced to assuming that an unnatural irritability of the nerves—either general and combined with hysteria, or else local and limited to the genital region—is the cause of the suffering (nervous dysmenorrhœa). But there is no doubt that we more often have to do with organic disease of the womb or ovaries or other pelvic organs, or with a fibromyoma, an infarction, a dislocation, or a degeneration of the ovaries. One form of dysmenorrhœal pain proceeds from spasmodic contractions which the womb sets up in order to expel some foreign body from its cavity—a polypus, a blood-clot, or a collection of liquid blood. The obstacles to the menstrual outflow are stricture of the cervical canal, either congenital or acquired, acute flexion of the womb, and growths blocking up the channel ; perhaps also spasm of the internal os (obstructive dysmenorrhœa). In this form the pains present a characteristic intermitting type, like those of colic or like labor-pains, the spasms corresponding with the periods of uterine contraction (uterine colic); and these attacks, which are more severe before the flow begins, subside after the contractions have expelled the stagnant blood from the uterine cavity.

Membranous dysmenorrhœa is a special form of obstructive dysmenorrhœa. Under normal conditions at the period of denidation, the outer layers of the proliferating mucous lining of the womb undergo fatty degeneration, and are cast off imperceptibly and in minute particles. Sometimes, however, by a morbid exaggeration of this process, there arises an exfoliation more or less profound of the mucous membrane, which then comes away in sheets. Indeed, the entire menstrual decidua may be cast off, in the form of a complete three-cornered sac corresponding to the form of the uterine cavity, with its smooth inner surface riddled by holes (mouths of the uterine glands), and its outer surface rough and ragged. Microscopic examination of the connective tissue of the mucous membrane, and the finding of the glands embedded in it, will prevent confusion of such an object with a mere fibrinous clot ; while the more difficult distinction between this process and that of an early abortion will be aided by the circumstance that the expulsion of menstrual decidua is seldom confined to a single menstruation, but is often repeated for months and years, even up to the climacteric period.

The chronic metritis and endometritis which often accompany this form of exfoliative menstruation are regarded by many as the cause of the process. The affection is attended by colic of greater

or less violence, according as the expulsion of the membrane and blood is retarded or accelerated. The diagnosis depends upon discovery of the membrane.

The prognosis and treatment of dysmenorrhœa must of course depend mainly upon a true understanding of its cause. Obstructive dysmenorrhœa is best treated by artificial widening of the cervical canal, either by bloodless dilatation, or else by incision or divulsion, which give far better results. For details we refer to the text-books on surgery. In membranous dysmenorrhœa, the objects of treatment should be the metritis and endometritis, regarded by some as the causes of the symptoms. When no complication forbids it, after subsidence of the period we may resort to applications of nitrate of silver, liquor ferri sesquichloridi, carbolic acid, or tincture of iodine in the cavity of the uterus, always observing the precautions given elsewhere. Although the result will probably not be perfect, yet a thinning or temporary arrest of the membranous formation may be obtained. Here, too, in obstinate cases, dilatation of the uterine neck may facilitate the escape of the decidua, and thus diminish the uterine colic. Violent pain is to be allayed by opiates, either by the mouth, rectum, or hypodermic injection, and by hot compresses to the belly. If there be fever, and the pain assume an inflammatory type, an acute metritis or perimetritis is to be feared, and proper steps should be taken to meet it.]

CHAPTER X.

RETROUTERINE HÆMATOCELE—HÆMATOMA RETROUTERINUM—PELVIC HÆMATOCELE.

[The French were the first, and *Nélaton* was the foremost, who investigated the subject of retrouterine hæmatocele, and fixed its position as a form of disease. The seat of a hæmatoma may be either within or without the peritoneal sac. The latter is very rare, occurring from hæmorrhages into the areolar tissue surrounding the cervix and vagina, and into that between the folds of the broad ligaments of the womb. These extravasations do not present any very definite symptoms, but merely form a tumor of variable characteristics. As regards the intraperitoneal hæmatoma, of which alone we now treat, let us premise that it is not every free accumulation of blood in the peritoneal sac that is included under this term; for a free collection of blood, like a free effusion of serum, cannot at first present to the touch a well-defined tumor. We limit

the term to those bloody effusions which are cut off from the peritoneal sac above them, and which are completely enclosed by inflammatory exudation and adhesion, so that they may exhibit all the qualities of a tumor. It is true that a free extravasation, after the blood has coagulated, may present to the examiner's touch an object of a certain solidity and firmness; but it does not offer the resistance opposed by the tensely-stretched walls of the encapsulated blood-tumor, into the cavity of which more and more blood is escaping, nor does it displace the neighboring organs. The effused blood usually collects in the most dependent part of the peritoneal cavity, the deep pouch between womb and rectum known as Douglas's cul-de-sac. It pushes the womb forward and upward, and the rectum backward. Douglas's fold is forced downward upon the posterior vaginal fornix, thus forming a large tumor, which may even extend upward above the brim of the pelvis.

An anteuterine hæmatocele, which is a very great rarity, consists in an encapsulated blood-tumor situated in the shallow anterior pouch between the bladder and the womb. An intraperitoneal hæmatoma may form in one of two ways: Either the hæmorrhage takes place within a closed pouch already formed by previous peritonitic adhesions, or else the bleeding may come first, and the encapsulation follow as a result of adhesive peritonitis which the contact of the effused blood excites in the peritonæum of the superimposed coils of intestine. The capsule once closed, a further extravasation may distend the sac and gradually displace the neighboring organs.

The sources of pelvic hæmorrhage are many and diverse, and during life they can, as a rule, only be conjectured. The bleeding proceeds most often from the ovarian vessels, and the insignificant physiological loss of blood which accompanies rupture of a Graafian vesicle may be aggravated into a profuse and alarming hæmorrhage. This may take place as a consequence of an excessive hyperæmia of the sexual organs, caused by violent exertion, or by blows, falls, mental excitement, masturbation, or coitus. Various diseases of the ovary, such as inflammation with abscess, bursting of a hæmorrhagic cyst, or rupture of a superficial varicose vessel, may have a like effect. In certain disorders of the tubes, so large an accumulation of blood or of inflammatory exudation may form in them as to result in rupture or perforation of their walls. It is less probable that the blood can accumulate in the tubes (as it does in the womb in hæmometra), and thence can pass through the abdominal opening into the peritonæum in sufficient quantity to form a tumor. It has long been known that by the bursting of a tubal or extrauterine pregnancy, if the resulting hæmorrhage is not speedily fatal, a hæmatoma

may form. Rupture of varicose veins of the broad ligaments may lead to intra- or extra-peritoneal hæmatoma, according as the blood passes into the pelvic cavity or into the areolar tissue between the serous folds. Finally, the pelvic peritonæum itself is a not unfrequent source of the bleeding. *Dolbeau* first announced that the blood might flow from vessels in a pseudomembrane, the product of a slow peritonitis. *Virchow* shares his views, which are in some measure supported by the fact that in a slighter degree a similar process may take place in men. The predisposing factors in pelvic hæmorrhage are the catamenial periods, the disorders giving rise to the so-called hæmorrhagic diathesis, scurvy, purpura, icterus gravis, and the hæmorrhagic exanthemata. Gynecologists differ greatly as to the frequence of the affection. *Schroeder* found it in five per cent. and *Seyfert* in seven per cent. of his cases. *Beigel* thinks that mild cases are very common, but that they run their course undetected except by accident. The disease is liable to arise at any time throughout the whole period of sexual activity; it is most common between the ages of twenty-five and forty, and in women who have borne children.

The formation of a pelvic hæmatocele is usually preceded by symptoms arising from the remoter fundamental disturbance which causes the hæmorrhage. The establishment of the hæmatoma itself is in most cases attended by unmistakable signs of internal bleeding, sudden pain in the belly, faintness, pallor, feeble pulse, coldness of the extremities, nausea, vomiting, and the like. Sometimes these symptoms recur again and again, whenever, as may happen from time to time, the bleeding is renewed. The history of an obscure case of this kind may furnish important data toward forming a diagnosis. The hæmorrhage having taken place, there ensues a more or less severe peritonitis, excited by the presence of the effused blood. This has the effect of encapsulating the effusion, unless indeed it be already included within an envelope, the result of ancient adhesions. Then follow the symptoms of pressure and crowding of neighboring organs by the tumor, tenesmus or impediment at stool, vesical pressure, and neuralgia from pressure upon the nerves of the lower extremities or engorgement of the dislocated womb, sometimes of such intensity as to cause metrorrhagia. The most important diagnostic test is by vaginal and rectal touch, whereby we may detect a retrouterine tumor of rapid growth, which often rises above the pelvic brim, but which also bulges downward into Douglas's culde-sac, and more or less against the posterior vaginal wall. The tumor is soft, elastic, even fluctuating to the touch, but grows more firm after a few days, when the blood has coagulated. Should re-

absorption take place as the weeks or months go by, it becomes very hard and uneven, and at the same time shrinks, becoming smaller and smaller, until it finally vanishes altogether, save a small lump close to the back of the womb. In unfavorable cases, as when the hæmatoma is very large, the size and tension of the tumor continue to increase ; the fever, the tenderness upon pressure, and other signs of local peritonitis are aggravated ; so that a distinction between an inflammatory exudation and a hæmatocele may at this period be very difficult, unless the sudden onset of the attack and the precursory signs of internal hæmorrhage can be taken into account. In such cases the blood is generally discharged spontaneously through the rectum or vagina, and the sac may finally close up after gradually evacuating its black, grumous, tarry contents. In exceptional cases a hæmatoma proves fatal through sloughing of the sac-wall and blood-poisoning, or through general peritonitis after bursting of the sac into the abdominal cavity ; for it would seem that the altered blood is far more irritating to the peritonæum than that which is recently effused. Long-continued suppuration of a fistulous sac may finally wear out the patient.

The treatment of a hæmatoma requires, above all, perfect and continued rest, and avoidance of anything which can accelerate the circulation. If the bleeding persists or recurs, we must apply ice to the abdomen or insert it into the vagina, at the same time giving the liquor ferri sesquichloridi internally, and injecting ergotin beneath the skin. Should the symptoms of hæmorrhage threaten life itself, transfusion of blood or milk is indicated, as in all other hæmorrhages. Pain is best allayed by hypodermics of morphine. The inflammatory symptoms and local peritonitis are to be met by striving, by full doses of opium, to arrest movement of the bowels, and thus to favor the adhesions by which the hæmatoma is becoming enclosed. Only after the inflammation has abated should the bowels be cautiously moved by enemata or by irrigation (*Hegar's* method). Gynecologists differ as to the merit of evacuation of a hæmatoma by puncture. Since opening the sac entails the danger not only of renewal of the bleeding, but also of putrefaction within the sac and its consequences, it should not be lightly attempted, nor until all hope seems lost of resorption, or else until violent symptoms make it seem hazardous to await spontaneous evacuation. The sac is to be punctured with a small trocar—or, better still, by the aspirating needle—the entrance of any air being carefully prevented. Puncture is most commonly made through the vagina or rectum. Should the contents prove to be suppurating or putrid, the opening must be freely enlarged, and the putrescent matter as

completely as possible removed by disinfectant irrigation, in order to prevent the consequences of septic absorption.

When the symptoms of a hæmatoma are mild, and it seems inclined toward absorption, the patient is merely to be kept at rest, with a light, nutritious diet, and impediments to the stools or urine are to be properly relieved.]

SECTION III.

DISEASES OF THE VAGINA.

In this section we shall only treat of the inflammations of the vagina, leaving the congenital anomalies, tumors, and other diseases of that organ, to the text-books on obstetrics and surgery.

CHAPTER I.

VIRULENT CATARRH OF THE VAGINA.

Etiology.—For the pathogeny and etiology of gonorrhœal catarrh of the vagina, we may refer to what we have said of virulent catarrh of the male urethra. Anatomically, it is not distinguishable from non-virulent catarrh, but its course and origin are different. The disease is not induced by sexual excess, or any other cause than infection with gonorrhœal matter.

Anatomical Appearances.—In women the chief seat of virulent catarrh is the mucous membrane of the vulva and vagina; more rarely it extends to that of the uterus. But in almost all cases the catarrh extends to the urethra, and this is important in the diagnosis. The affected mucous membrane at first shows the changes peculiar to the most severe form of acute catarrh; subsequently those of chronic catarrh. The secretion, which, in the beginning, is usually scanty, subsequently becomes very copious, purulent, irritates the vulva and inner surface of the thighs, but is only peculiar in that it is the bearer of the contagion.

Symptoms and Course.—The first symptoms of gonorrhœa in the female, a feeling of itching and warmth in the sexual organs, and a scanty mucous discharge, are not very characteristic, and are often unnoticed. A few days after the commencement of the disease, there are severe burning pain in the genitals, swelling of the vulva, *ardor urinæ;* but these troubles rarely become so severe as to interfere greatly with

walking, sitting, and moving the body. The secretion, which in this stage is yellowish-green, thick, and purulent, often oozes up in large quantities between the labia; in the vulva and its vicinity, even to the anus, we find shallow ulcers, which are not to be confounded with chancres. We may almost always press pus out of the urethra. After the disease has lasted a fortnight or three weeks, the pain abates and ceases; the discharge loses its purulent appearance, but continues a long while till the secretion, which constantly becomes more mucous, dries up, and loses its power of inoculation.

Treatment.—The local treatment, which we preferred to internal remedies for gonorrhœa in men, is almost exclusively used for the disease in women. Considering the different seats of gonorrhœa in the two sexes, it may be readily understood that copaiba or cubebs, whose active constituents are excreted with the urine, may have an effect on gonorrhœa in the male urethra that it cannot have on the disease in the female, where the vagina is chiefly affected. While there is severe pain, we prescribe scanty diet, laxatives, long-continued sitz-baths, during which a speculum should be left in the vagina, if its introduction be not too painful. If there be no symptoms of inflammation, or if they have been allayed, we may employ injections of solutions of tannin, nitrate of silver, alum, sulphate of zinc, acetate of lead, etc. Injections of plumbi acet. crystall. ʒ iij.—Aquæ conj. j, as recommended by *Ricord*, are very efficacious; instead of being injected, this solution may be poured in through a speculum, and the latter slowly withdrawn, so that the fluid shall come in contact with all parts of the vagina. In very obstinate cases we may introduce wads of charpie, sprinkled with alum, into the vagina, or touch the parts with solid nitrate of silver.

CHAPTER II.

NON-VIRULENT CATARRH OF THE VAGINA.

Etiology.—*Kölliker* and *Scanzoni*, who have carefully examined the secretion from the vaginal mucous membrane, both in health and disease, found perfectly healthy secretion in very few women, and only in those who had had no children, and had not frequently indulged in coitus. It was so scanty that the surface of the mucous membrane was only lubricated by it; it was nearly as clear as water, fluid, only viscid, white or yellowish in spots; it was almost always acid, and, besides a small amount of pavement epithelium, it contained no noticeable solid constituents. Shortly before and after menstruation the secretion was more copious, always fluid, and almost always acid; at this time it

contained more epithelial cells, and, after the cessation of the menses, as long as it retained its red color, there were more or less blood-corpuscles in it. In catarrhal affections they found the secretion either milky and thin, or more yellowish and thicker. The more opaque, white, or yellow, the secretion appeared, the more numerous were its pavement epithelium and young cells (mucous and pus-corpuscles), often also the infusoria discovered by *Donné* (*trichomonas vaginalis*), and some few fungous filaments and vibriones. Hence we see that these observers rarely found a perfectly normal secretion, and that catarrh of the vagina is one of the most common of female diseases. Irritation of the vagina, by sexual excess, is the chief cause of catarrh of that part, it is true; but it is often induced by other causes, which were mentioned when speaking of uterine catarrh. It is particularly to be borne in mind that catarrh of the vagina, like that of the uterus, depends as often on constitutional as on local causes. A consideration of the exciting causes shows that this disease must be rare during childhood; and this is true, except in the cases where oxyuris have passed over the perinæum from the anus to the vagina, and have there caused great irritation.

Anatomical Appearances.—In *acute* catarrh we find the mucous membrane bright red, swollen and relaxed; in many cases we see small prominences on it, which give the membrane a granular appearance, and which are not, as was formerly supposed, due to distention of the follicles, but to swelling of the mucous papillæ. Sometimes these changes affect the entire vagina, at others only parts of it. At first the secretion is scanty, but, after the disease has lasted a very short time, it becomes more copious and more or less opaque.

[The inflammation usually spreads all over the vagina, and over the vulva as well. Sometimes, however, a portion only of the vagina is involved. For instance, at the beginning of a vaginal gonorrhœa the lower part only will be attacked, while a catarrh caused by disease of the cervix or uterus will be confined chiefly to the upper part of the canal.

Hildebrand describes a peculiar ulcerous form of inflammation of the upper vagina, caused by loss of the epithelium and slight hypertrophy of the papillæ, and finally adhesion of the vaginal portion to the vaginal walls, so that the fornix vaginæ is obliterated, and the os uteri is to be felt at the top of a funnel-shaped tube. *Schroeder* has also seen this disease, which in one case resulted in complete atresia, in another in partial closure of the vagina below the vaginal portion, and in a third case in complete contraction (stenosis) of the entire upper vaginal region.]

In *chronic* catarrh the walls of the vagina appear distensible and

flaccid. The membrane is more bluish red and its surface more frequently granular than in the acute variety; the secretion is sometimes more milky, sometimes more yellow and thick. Not unfrequently the relaxation of the vagina induces prolapse, particularly of the anterior wall.

Symptoms and Course.—In non-virulent catarrh also, if it begin acutely, the patient complains of prickling and burning in the private parts; but, as the mucous membrane of the urethra is unaffected, she has no pain on urination. In chronic catarrh the discharge of the abnormal secretion, the leucorrhœa (*fluor albus vaginalis*), is often the only symptom of the disease. The chief means of deciding whether the discharge be from the vagina or uterus is by examination with the speculum. Many women bear the drain without any injury; in others it induces anæmia, pallidity, weakness, and emaciation.

Treatment.—In treating non-virulent vaginal catarrh, just as in uterine catarrh, we must first attend to the causal indications. But we cannot often cure the disease without accompanying local treatment. We would chiefly recommend the hip-baths and injections mentioned in the last chapter; in obstinate cases, the introduction of a tampon sprinkled with alum, and even the application of solid nitrate of silver.

CHAPTER III.

CROUPOUS AND DIPHTHERITIC INFLAMMATION OF THE VAGINA.

Croupous and diphtheritic inflammations of the vaginal mucous membrane are common during puerperal fever, but are rare except in the puerperal state. They either depend on local irritation or on constitutional disease. Thus, the discharge from a sloughing cancer of the uterus, the urine constantly flowing through a vesico-vaginal fistula, a bad pessary, or a large uterine polypus projecting into the vagina, may induce croupous or diphtheritic inflammation of the vaginal mucous membrane; while the disease is also often observed in the later stages of typhus, cholera, measles, and small-pox, and accompanying similar affections of other mucous membranes. Usually only certain spots of the vagina are covered with croup-membrane, or changed to diphtheritic sloughs. In the vicinity of these spots the mucous membrane is dark red; after the slough has been thrown off, irregular losses of substance remain; these are sometimes superficial, at others deep. Our attention is called to the disease by severe pain in the parts, and by a fetid, often bloody

discharge, which begins in a few days. Local examination is the only means of certain diagnosis. Where a large or putrefied pessary has caused the inflammation, its removal, and the use of lukewarm, and later of cold and astringent, injections, ordinarily suffice to cure the disease. The affection usually disappears readily also if a large polypus, that has forcibly distended the vagina, be removed. On the other hand, where sloughing cancer or vesico-vaginal fistula has caused the disease, we must limit ourselves to a palliative treatment. In those cases occurring in the course of infectious diseases, it is usually sufficient to attend to cleanliness, as the secondary disease generally passes off with the primary.

DISEASES OF THE NERVOUS SYSTEM.

SECTION I.

DISEASES OF THE BRAIN.

CHAPTER I.

HYPERÆMIA OF THE BRAIN AND ITS MEMBRANES.

Etiology.—For a time the fact was ignored that on autopsy the blood-vessels within the skull were sometimes found distended and at others empty, and it was supposed that the amount of blood contained in the closed cranium of an adult could neither increase nor diminish, but was constant; and that anæmia or hyperæmia was only supposable when the brain-substance was increased or diminished, that is, when there was hypertrophy or atrophy of the brain. This view was based on the following reasoning: The brain is not compressible, at least not by the pressure to which it is subjected from the contents of the blood-vessels; and it is surrounded by walls which do not expand; consequently only the same amount of blood can enter the skull as passes out from it, and conversely only as much blood can pass out of the skull as enters it. This reasoning is false, as it starts with the supposition that the contents of the cranium consist only of the membranes of the brain, the brain-substance and the blood-vessels with their contents; it leaves the cerebrospinal fluid out of consideration. This, which is a simple transudation, can rapidly increase or diminish, and can at least partly pass into the spinal canal, which is not entirely enclosed by rigid walls. In almost all autopsies it may be seen that the amount of blood contained in the vessels and the amount of cerebrospinal fluid are in inverse proportion; that a distention of the vessels of the meninges is accompanied by a decrease of arachnoid fluid, and conversely that, when the vessels are less full, the meshes of the

textus cellulosus subarachnoidealis contain a greater amount of serum. Only when the brain is atrophied do we find œdema of the membranes with overfilling of the vessels; and only when an effusion of blood, a tumor, or a collection of fluid in the ventricles, has contracted the space in the skull do we find, along with anæmia, dryness of the membranes, and disappearance of the sulci between the cerebral convolutions.

The division of cerebral hyperæmia into active and passive, or, to retain the expressions previously used, into fluxionary and congestive, is practically valuable not only on account of the consideration being easier, but because the symptoms of one form differ from those of another.

Fluxionary hyperæmia results—1. From increased heart-action. It is true that, in this case, while the arteries are fuller than usual, the veins are less so; hence the entire amount of blood in the vessels of the brain and its membranes is not increased by the stronger action of the heart. But the increased lateral pressure induces increased fulness of the capillaries, and it depends chiefly on these (not on the amount of blood in the large vessels) whether or not the brain acts normally and is normally nourished. This form of cerebral hyperæmia occurs temporarily from augmented energy of contraction of a healthy heart, as in fever and great bodily or mental excitement; it is habitual in the permanently increased activity of a hypertrophied heart, but only when the hypertrophy is an independent disease, or in case it accompanies an obstruction to circulation, when it has become greater than is necessary for the compensation. Simple, non-complicated hypertrophy of the heart is not frequent, and occurs almost exclusively in topers and persons who continually do hard work. On the other hand, hypertrophy that has become greater than was necessary to compensate the obstruction to the circulation is quite frequent. Examples of this are the occasional enormous hypertrophies of the left ventricle when there is insufficience of the aortic valves, and perhaps also the hypertrophy of the heart in morbus Brightii.

2. Fluxionary hyperæmia of the brain results from too slight resistant power of the afferent blood-vessels, whether this be congenital or acquired. When the cerebral arteries have delicate, thin walls, so that they yield to an increased pressure of the blood sooner than the other arteries of the body do, and hence, when the action of the heart is only moderately increased, fluxionary hyperæmia of the brain is induced, it is customary to say that the person so affected has a tendency to "rush of blood to the head."

3. Fluxionary hyperæmia to the brain results from an increase of the lateral pressure in the carotids as a consequence of obstructed escape of blood from the aorta into other branches. As a type of this

"collateral fluxion" to the brain, we may mention the habitual cerebral hyperæmia where there is contraction or closure of the aorta at the point where the arterial duct terminates in the aorta (Volume I., page 363). It frequently results from compression of the abdominal aorta and its branches, by the distended intestines and by exudations. In the same way obstruction of the cutaneous circulation during the cold stage of intermittent fever, and from the action of severe cold, induces collateral fluxion to the brain. According to *Watson*, in cold nights many unfortunates are arrested in the streets for being drunk, when they are only suffering from cerebral hyperæmia as a result of disturbance of the cutaneous circulation. It is not improbable that severe muscular exertion may also induce increased fulness of the carotids and fluxionary cerebral hyperæmia, by the pressure of the contracted muscles on the capillaries.

4. A fourth cause is paralysis of the vasomotor nerves of the cerebral vessels. Physiological experiments show that, if the cervical portion of the sympathetic nerve be divided, the vessels on the corresponding side of the head become dilated. The cerebral vessels appear to be similarly affected by the use of spirituous liquors, by some poisons, as well as by great emotions and excessive mental activity. I would particularly call attention to the last cause, as I have frequently seen dangerous hyperæmia of the brain after too prolonged mental labor, which resulted fatally from the occurrence of œdema. We can hardly give any other explanation for these cases, than that the walls of the vessels are paralyzed by the above influences, their calibre dilated, and the supply of blood consequently increased.

5. Lastly, fluxion to the brain results from atrophy of that organ. The space left in the skull by the disappearance of the brain-substance is partly filled by the dilatation of the vessels. We shall find this dilatation of the vessels to be a frequent cause of their rupture; and, as atrophy of the brain often follows apoplexy, it is also a cause of returns of apoplexy. We shall not attempt to say whether or not the fluxionary hyperæmia of the brain, occasionally observed during convalescence from severe disease, is the result of atrophy of the substance of the brain or of the neuroglia and consequent dilatation of the vessels.

Passive hyperæmia, congestion of blood in the brain, depends—

1. On compression of the jugular veins and vena cava descendens. As a type of this form, we may mention the excessive cerebral congestion caused by strangulation. The jugular veins are most frequently compressed by enlarged thyroid or lymphatic glands, the vena cava descendens by aneurisms of the aorta.

2. Congestion of the brain results from energetic expiratory move-

ments while the glottis is contracted. In coughing, straining, playing wind-instruments, etc., as we have often shown, the flow of blood into the thorax is hindered; the pulmonary circulation contains too little, the general circulation too much blood. Under such circumstances, the brain must suffer far more from hyperæmia than other organs supplied by the general circulation, particularly the liver, spleen, and kidneys; because the passage of blood through the upper aperture of the thorax is more impeded than it is through the lower, since these abdominal glands undergo the same compression from the abdominal muscles that the veins of the thorax and the heart do.

3. We have cerebral congestion in all those diseases of the heart where the function of that organ is impaired, if they are not complicated by other anomalies of opposite effect, and so compensated. In non-compensated valvular disease of the left ventricle, the whole amount of blood in the vessels of the brain and its membranes is not increased, it is true, since, while the veins are overfilled, the arteries are less full; but the overfilling of the veins obstructs the flow of blood from the capillaries, thus inducing capillary hyperæmia, which, as we have shown, is the most important cause of the cerebral hyperæmia. The affection caused by valvular disease of the right heart is far greater than that from valvular disease of the left heart; for, in the former case, not only is the escape of venous blood from the brain impeded, but the entire amount of blood in the skull is increased.

4. The same state of affairs occurs in extensive compression or atrophy of the vessels of the lungs as in pleuritic effusions, emphysema, or cirrhosis of the lungs. In these diseases also, when the right ventricle is not hypertrophied in proportion to the impediment to the circulation, the systemic circulation is overloaded at the expense of the pulmonary, and the amount of blood in the skull is increased. Since the contents of the cerebral veins and sinuses cannot pass into the overfilled jugular veins, the same excessive cyanosis occurs in the brain that is so evident in the skin, and which is almost pathognomonic of the diseases in question. The last stage of emphysema gives us an excellent opportunity of studying the gradual development, steady increase, and, finally, the severest symptoms of congestion of the brain.

Lastly, we must mention a form of cerebral hyperæmia which is neither fluxionary nor congestive, and which is peculiarly important, as it should be very carefully avoided by persons suffering from disease of the blood-vessels, and having a tendency to apoplexy; I mean those cases of hyperæmia that appear as one symptom of temporary general plethora induced by a very free supply of food and drink.

Anatomical Appearances.—On *post-mortem* examination, it is often difficult to decide whether the vessels of the cerebral membranes

and still more so whether those of the cerebral substance, have been more than normally filled with blood during life. Mistakes in the account of the autopsy are frequent.

The mistakes regarding the amount of blood in the cerebral membranes depend partly on the fact that, when unaccustomed observers find the vessels much distended at the dependent parts of the surface of the brain, they diagnose a hyperæmia of the cerebral membranes, even if the blood has only sunk downward, and the vessels in the upper part be empty. Still more frequently another error is committed even by practised observers: that is, from a similar distention of the vessels on the convex surface of the cerebrum, hyperæmia of the cerebral membranes is decided on without looking further. It should be borne in mind that the arteries supplying the cerebral membranes with blood lie at the base of the brain, and that only very fine arterial twigs reach the convexity of the greater hemispheres. All the large blood-vessels usually seen on the surface of the brain, when the skull is opened, are veins. Distention of these veins is a normal appearance, if the individual has died of an acute disease by which his blood was not consumed, or, if he has died suddenly from suffocation, acute poisoning, or from some other accident involving no loss of blood. Hence it is entirely wrong, in such cases, to decide, from the overfilling of the veins, that there has been hyperæmia of the brain or its membranes during life, and to connect this pretended hyperæmia with the symptoms that have been observed. In the history of the *post mortem*, accounts of excessive hyperæmia of the brain and its membranes are often combined with others of a similar excessive hyperæmia of the lungs, liver, kidneys, etc. If there were no mistake here, did we not have to suppose that, in the body of a previously healthy person, who had died neither from exhausting disease nor loss of blood, the normal amount of blood was often considered pathological, these accounts of *post mortem* would only convey the absurd idea that the entire amount of blood in the body was increased by poisoning, suffocation, etc. We must make it a rule to consider hyperæmia of the cerebral membranes as proved only in those cases where the finest vessels also are injected, and where the overloading of the cerebral vessels is not at all in proportion to the amount of blood in other organs.

The great difficulty of detecting, in the cadaver, a hyperæmia of the substance of the brain, that has existed during life, depends chiefly on the fact that the first fine ramifications of the vessels supplying the brain take place in the pia mater, and that the vessels passing thence to the substance of the brain are mostly capillary (*Luschka*). As these can scarcely be recognized with the naked eye, it is customary to use the size and number of the drops of blood oozing up on a cut

surface of the brain as a means of determining the amount of blood contained in the cerebral vessels. I do not deny that this sign has some value, particularly in judging of passive cerebral hyperæmia; but I must add that the size and number of these drops of blood depend far more on the fluidity of the blood than on the fulness of the vessels. At all events, in cases where there can be no doubt of death having resulted from increased flow of blood to the head, or from its obstructed escape thence, on a section through the brain, its substance is often found very pale, and on its cut surface only a few small blood-drops ooze up. This circumstance, and the symptoms of paralysis occurring in the severer cases of cerebral hyperæmia (of which we shall speak more fully in the next paragraph), cause it to appear to me very probable that, when there is increased lateral pressure in the small arteries and veins of the brain, a transudation of serum from them into the perivascular spaces and interstices of the brain may very readily take place, and cause compression of the capillaries. It is only in yielding and distensible organs and tissues, which are not enclosed by firm envelopes, that any considerable œdema can coexist with a normal fulness of the capillaries. In all tissues enclosed by fascia or other firm capsule, œdema causes anæmia of the capillaries. If the size of the brain be not diminished by atrophy, and if the skull be closed, or, it remaining opened, if the dura mater be tense, there is no doubt that a slight transudation of serum will suffice to completely compress the capillaries of the brain. It is true, we cannot be sure, from *post-mortem* examination, that there is such a secondary œdema; but the supposition that such is the case appears to us perfectly justifiable when a patient has died with the symptoms of cerebral paralysis, and if, on autopsy, we find that the very white hue of the brain-substance and the slight number of small blood-points appearing on its cut surface contrast strongly with the distention of the large vessels in the meninges.

Where the hyperæmia is often repeated, atrophy of the brain and decided dilatation of the vessels result. The vessels of the meninges, which are unmistakably dilated, run a tortuous course; on section through the brain, we may distinctly see the gaping mouths of the vessels; the dilatation may even be observed in the capillaries on microscopical examination. There is plenty of serum in the sub-arachnoid spaces, the brain-substance is moist and shining. This appearance, which is frequent in topers, is readily understood, if we remember that, in atrophy of the brain, the fluid contents of the skull must increase so as to fill the cavity. It is doubtful whether the development of the Pacchionian bodies can also be considered as a result of repeated hyperæmia; they are sometimes solitary, sometimes

grouped as whitish, opaque excrescences of the arachnoid, coming particularly along the sides of the longitudinal sinus. The pressure that they exercise on the dura mater separates its filaments, so that they perforate it; by further pressure they also cause atrophy of the bone, and they are then found embedded in little fossæ in the skull. Microscopically, they consist of connective tissue; occasionally they contain fat and chalky salts.

Symptoms and Course.—Before taking up the symptomatology of hyperæmia of the brain, I shall warn against the wide-spread error, so injurious to the patient, of considering all cases of disturbance of function of the brain, where severe structural changes can be excluded, as due to hyperæmia (or anæmia).

Thus, the disturbance of the cerebral functions in fever is not due to increased afflux of blood to the brain from excited action of the heart; but, as we have repeatedly pointed out, it depends partly on the high temperature of the blood in the cerebral vessels, partly on its abnormal quality, the "feverish state," a necessary result of the increased transformation of tissue during the fever. Delirium and other severe cerebral troubles are most common in the so-called asthenic fevers, just where the increase of bodily temperature and the production of warmth attain the highest grade, while the heart's action is hastened, but weakened, and there is no fluxion to the brain.

According to the observations made during the last war, as well as from the valuable investigations and experiments of *Obernier*, the symptoms of *sun-stroke*, or *insolatio*, do not, as was formerly supposed, depend on hyperæmia of the brain, induced by the action of the sun's rays on the head. The symptoms of this disease consist in a paralysis of all the functions of the brain, occurring either suddenly or gradually. In the latter case the paralysis is preceded by excitement, delirium, and other symptoms of cerebral irritation. It has been determined that, in our zone at least, the action of the sun's hot rays is not alone sufficient to induce these severe attacks, but that they only occur when individuals are subjected to great fatigue, on a very hot day, particularly if, at the same time, they sweat very little, because they do not drink enough. We may assume that, under such circumstances, while the radiation of heat is limited on account of the high temperature of the surrounding atmosphere, while the production of heat is increased by the active muscular exercise, and the coolness induced by free perspiration is limited, there is an overheating of the body, an increase of the bodily temperature to a height incompatible with life. We have already sufficiently explained the significance of the fulness of the veins of the meninges, which is found on autopsy in cases of sun-stroke.

[The condition known under the term "sunstroke," or better, "heatstroke" (for the direct action of the sun's rays is by no means essential to it), used formerly to be ascribed to a hyperæmia of the brain; a belief which has now been completely exploded by experiments upon animals and by post-mortem examinations. It is now known, from the researches of *Obernier*, that in this disease there is a serious derangement of the heat-producing function and a great rise in the bodily temperature, which in extreme cases may reach 109° or 110° Fahr. Absolutely nothing as yet is known, however, of the anatomical lesions upon which sunstroke depends. It was formerly ascribed to grave alterations in the blood, to its over-acidity by overloading with carbonic and lactic acid, to destruction of its red corpuscles, to accumulations in it of urea, and the like. *Koester* in one case has found a hæmorrhage in the right superior sympathetic ganglion, and in the right sympathetic itself, as well as smaller extravasations about and in the two vagi, and thinks we should look for the derangements in the vasomotor and respiratory nervous systems. *Arndt* seeks for an anatomical basis for the disease in the so-called "turbid swelling" (*trüben Schwellung*) developed in the brain, liver, kidneys, and other tissues, under the influence of the enormously high temperature, just as it occurs in high fever from other causes. The different symptoms presented during life may depend upon the degree in which the turbid swelling has attacked individual organs. In milder cases the temperature is below 104°; the mind is muddled, the chest oppressed and uneasy, the pulse very frequent, the heat great, the skin moist, and there is a general but moderate feeling of languor. If relief cannot be gained through water-drinking and quiet, graver symptoms appear. The temperature then rises above 104°, the pulse to 130 or 140; the sweating becomes profuse and the thirst distressing. The patient loses control over himself, and is oppressed by fulness of the head, dizziness, sensory delusions, and precordial distress. He is forced to sit down, or else sinks suddenly unconscious, and lies with flickering imperceptible pulse and sunken features. Sometimes there are epileptiform fits. Although under proper treatment many very severe cases recover, yet the number which end fatally, particularly during the marches of armies in hot weather, is sometimes large. Many patients improve at first, and then relapse unexpectedly. This fact, as well as the experience that recovery after sunstroke is slow, that it has many sequelæ, such as vague pains, disposition to faint, indigestion, unnatural drowsiness, and the like, which are often obstinate, and the circumstance that sometimes there remains a psychical irritability or a permanent impairment of intellectual function, all

indicate that the disorder has a definite material basis, which in bad cases is not always repaired.]

Lastly, the symptoms of acute alcoholic poisoning, as well as that from opium and other narcotics, do not depend at all, or at any rate depend to a very small extent, on over-fulness of the cerebral vessels, although in them the brain is hyperæmic. The case appears to be different in the symptoms induced by the immoderate use of liquor for a length of time, or the continuous misuse of narcotics, a practice which has greatly increased since the introduction of subcutaneous injections of morphine. In such cases cerebral hyperæmia plays a more important part, at least, than it does in the symptoms of intoxication and of acute opium-poisoning.

[Abnormal fulness of the brain and its membranes is a common enough affection. Its symptoms may vary greatly. Not unfrequently they are so insignificant as to attract little or no attention; so that many persons with habitual plethora about the head are familiar with the sensation which it causes, and do not consult a physician. But there are other cases in which hyperæmia causes symptoms of the utmost gravity; and between the mere sense of giddiness after stooping which one man may feel, and the formidable—nay, fatal—apoplectiform seizure which may befall another, there is a long series of interchanging types, whose differences, however, by no means always correspond to differences in grade of the hyperæmia. Most of the symptoms bear the character of a morbid nervous excitement with exaltation, although others take on the form of depression and paresis. The psychical, the sensory, or the motor functions may be especially disturbed, according to the seat of the hyperæmia and to the receptivity of the individual. One might at first suppose that the symptoms of an active hyperæmia of the brain should be widely different from those of a passive congestion; since in the first there is an increased afflux to the brain of arterial blood, the constant supply of which is so essential to the continuance of the brain-function, whereas in the latter this arterial supply must be more or less diminished, and the symptoms of carbonic-acid poisoning should also be present. But in reality the two forms of hyperæmia bear a close resemblance; and this is due to the fact that a lack of blood in the brain is capable of causing like symptoms with those from overloading of the brain with arterial blood; and it is well known to the practitioner that the distinction between cerebral hyperæmia and cerebral anæmia may present obstacles at the bedside only to be surmounted by prolonged observation of the patient.

The symptoms of the mildest grade of hyperæmia of the brain consist in a feeling of pressure and weight in the head, or of an

actual headache, often accompanied by dizziness and an extraordinary susceptibility of the special senses. There is a buzzing in the ears, and a dazzling light before the eyes. This latter usually begins at a spot in the eccentric field of vision, with a quivering as though smoke were rising. It advances to the middle of the field, obscuring the whole, and renders accurate seeing impossible. The part first affected begins to clear, and in half an hour all is over. The arteries of the eyeball are branches of the internal carotid, and would naturally participate in an intracranial hyperæmia. Hence, in an obscure case the ophthalmoscope should aid us.]

Hyperæmia of the brain is marked partly by symptoms of increased excitability of the nerve-filaments and ganglion-cells and by their morbid excitement (symptoms of irritation), partly by symptoms of diminished or lost excitability of these nerve-elements (symptoms of depression). Usually the symptoms of irritation precede those of depression, in other cases the former do not occur, and the latter begin from the outset. It is commonly supposed that this difference of symptoms depends on difference of pressure on the brain from the more or less distended blood-vessels, and reference is made to the analogous action of the peripheral nerves, which are also irritated by a moderate pressure and paralyzed by a stronger one. This explanation appears very suitable as regards the symptoms of irritation. Experience shows that nerves, passing through bony canals in company with blood-vessels, are thrown into a state of increased excitability and morbid excitement by overfilling of these vessels; the nerve-elements of the brain, enclosed by the dura mater and skull, are in a like condition when the cerebral vessels are overfilled. On the other hand, the reference of the symptoms of paralysis to an increase of the "intravascular pressure" appears to me erroneous, for even in the severest forms of hyperæmia this pressure does not nearly approach the grade necessary to induce paralysis of a peripheral nerve. In support of the above view, it has been said that paralysis is also caused by the scarcely greater "extravascular pressure" of small extravasations on the brain; but I shall hereafter show that the apoplectic symptoms from cerebral hæmorrhage do not depend on contusion of the brain from the extravasation. It is much more probable that the symptoms of depression and paralysis arise because the requisite supply of arterial, oxygenated blood to the nerve-filaments and ganglion-cells of the brain is limited or entirely stopped in excessive cerebral hyperæmia. In congestive hyperæmia the escape of venous blood from the brain is checked; and it is evident that, when the veins finally become filled to a certain point, no new arterial blood can enter the capillaries. It is often asserted that the symptoms of cerebral hyperæmia are very similar to o

exactly identical with those of cerebral anæmia; this is true in regard to congestive hyperæmia and anæmia, and the explanation of the correspondence is easy. In both cases the brain lacks its new supply of arterial blood. To explain the symptoms of paralysis occurring in fluxionary hyperæmia also, we must take the hypothesis that, during its course, there is a secondary œdema of the brain, as a result of which we have capillary anæmia, a condition directly opposite to the original hyperæmia. The symptoms of cerebral hyperæmia are rendered more varied by the fact that, in different cases, the irritation or paralysis is more prominent at one time in the sensory, at another in the motory, and again in psychical functions. We cannot give any satisfactory explanation of this difference.

Among the symptoms of irritation in the *sensory* functions are headache, great sensitiveness to external impressions, and simple subjective impressions from the nerves of special sense. The headache, a very frequent symptom in all cerebral diseases, is very difficult to explain; we do not even know if it is of central origin (that is, if it originates in the parts of the brain where irritation causes symptoms of pain after the insensible greater hemispheres have been removed), or whether, as I think is more probable, it depends on irritation of the filaments of the trigeminus going to the dura mater. The great sensitiveness to impressions on the senses depends on the increase of excitability caused by the cerebral hyperæmia on the hyperæsthesia of those portions of the brain through which peripheral irritations are perceived. The patients do not exactly feel, see, and hear more sharply than ordinarily, but they are annoyed by irritations far weaker than such as usually annoy them. Light troubles them; a slight sound, or an insignificant irritation of the nerves of touch, excites disagreeable feelings. Morbid excitation (which must not be identified with increased excitability) of the same central parts causes the dazzling before the eyes, seeing sparks, roaring and buzzing in the ears, the sensation of formication, or of undefined pain, which are not induced by peripheral irritation. Among the *motory* symptoms of irritation we have restlessness, sudden starting, gnashing the teeth, crying out, without the expression of pain, the automatic movement of the extremities, twitching of single muscles, and lastly the general convulsions which are observed in the course of cerebral hyperæmia. In regard to the first of these symptoms, particularly the restlessness, the constant tossing around in bed, we may often be doubtful whether it depends on increased excitability of those portions of the brain through which the motor nerves and muscles are excited and motions induced, or on morbid states of excitement in the organs affected. On the other hand, there is no doubt that the twitching of individual muscles,

occurring independently of mental influences, and convulsions affecting the whole body, are the results of a morbid excitement of the cerebral motor centres. It is very remarkable that general convulsions occurring in cerebral hyperæmia and other affections of the brain, as well as in epilepsy and eclampsia, are not accompanied by other symptoms of cerebral irritation, but, on the contrary, are constantly combined with symptoms of cerebral paralysis, especially with complete loss of consciousness. By a series of valuable experiments, *Küssmaul* and *Tenner* have shown that the same combination of general convulsions, with loss of consciousness, may be induced by artificially obstructing all the arteries supplying the brain with blood; but it is entirely unexplained, and it is enigmatical, how it happens that the same pathological process induces a state of the greatest excitement in some parts of the brain and paralysis in others. Until this contradiction is explained, the investigations as to whether the above symptoms depend directly on the lack of oxygenated blood, or indirectly on changes occurring in the blood, which remains unrenovated on account of the obstruction, are of little value. Since these attacks of convulsions are constantly accompanied by symptoms of paralysis, and since we do not believe that the latter can proceed from the pressure of the distended vessels on the nerve-elements of the brain, and, lastly, since we may artificially induce loss of consciousness and general convulsions by preventing the flow of arterial blood to the brain, we consider the following decisions justifiable: The general convulsions and loss of consciousness occurring during hyperæmia of the brain, which are usually termed epileptiform attacks, either occur because in passive hyperæmia the veins become so full that more arterial blood cannot enter, or because the hyperæmia has induced a transudation of serum into the perivascular spaces and interstitial tissue of the brain, with consequent anæmia. Among the symptoms of psychical irritation, there is first such a rapid change and loose connection between the thoughts, that clear thinking is impossible. Along with this confusion of ideas, the patient often has false notions about himself and the outside world, or delirium.

Delirium is sometimes so real and intense that the patient cannot distinguish it from true perceptions. This is the origin of *hallucinations* and *illusions*. By the former we understand erroneous impressions, which are considered as the product of direct perceptions, without there being any actually existing external object corresponding to the impression. The patients think they see animals and other objects which are not present, and that they hear voices when all is quiet. By illusions, on the other hand, we understand the misapprehension or false interpretation of external objects which really exist.

As a result of illusion, the most harmless things may become objects of great terror to patients suffering from hyperæmia of the brain, particularly to children. *Dizziness*, which is one of the most frequent symptoms of hyperæmia of the brain, and of many other cerebral diseases, is, as a rule, a simple hallucination, since it consists in a vivid representation of a movement of the body of the patient himself, or of the bodies of surrounding objects which the patients imagine they see or feel, although they are actually at rest. We shall hereafter speak of other forms of dizziness. Closely related to the symptoms of increased mental excitement are sleeplessness and the disturbance of sleep by vivid dreams, symptoms which are very common in hyperæmia of the brain.

Among the symptoms of depression observed in the *sensibility* in cerebral hyperæmia, we must first mention a certain insensitiveness, a tolerance to external irritation, bright light, loud noise, etc. There is no reaction to slight irritation. On an increase of this in complete anæsthesia to complete cerebral anæsthesia, the patients do not perceive even the severest irritation. The excitability of the portions of the brain through which external impressions are perceived is lost. The *motor* symptoms of depression are decided slowness and sluggishness in the motions of the patients; "their limbs are as heavy as lead." If this state increases, there is complete inability to make any voluntary movement (cerebral paralysis). The excitability of the motor centres is extinguished. The *psychical* symptoms of depression are: loss of interest, and indifference; great slowness of thought, and limitation of the ideas; inclination to sleep, from which the patient is aroused with difficulty, and subsequently cannot be aroused at all. When this state is increased to the highest point, consciousness is completely lost.

In hyperæmia of the brain there is not unfrequently, also, a deviation from the normal in those movements that are excited by the cerebral nerves, independently of the will. Thus, in states of irritation the *pupil* is contracted, because the oculo-motor nerve is more excited; in depression it is dilated, because then the sympathetic filaments of the iris act more strongly. In the same way, just as, in physiological experiments, increased excitement of the vagus causes the heart to beat slower, while in central paralysis of the vagus, as well as after its division, the heart's action is more frequent. In paralytic states of the brain, respiration is often very slow, deep, and stertorous. Although we cannot give an exact explanation of this symptom, we may still call attention to the fact that, after division of the vagus in animals, besides other symptoms, there is always retardation of the respiration. Lastly, we must mention *vomiting* as a very frequent and important

symptom of irritation of the brain. Without understanding the process in the central organs, by which the motions of vomiting are brought about, we nevertheless know that it is artificially induced in animals by dividing the vagus, and then irritating the central end.

The above symptoms of morbid excitement, and of increased, diminished, or lost excitability of the brain, which we shall also meet in many other cerebral diseases, occur in the most varied groups in cerebral hyperæmia. When speaking of general convulsions, we called attention to the constant coincidence of this symptom, which is due to morbid increase of excitement in the medulla oblongata, with loss of consciousness, which indicates loss of excitability of the other central ganglion-cells and nerve-filaments; and we must add that, not unfrequently we meet other complications of symptoms of irritation with those of paralysis, in hyperæmia of the brain. For instance, the power of motion is occasionally lessened while the patient is under great mental excitement, complaining of headache, sparks before the eyes, etc. Hence, from the state of the pupil, the slowness or rapidity of the pulse, etc., we may recognize the state of excitement in which the central organs of the nerves governing these states are at the time, but we cannot come to any conclusion regarding the condition of other parts, or of the entire brain. The variety of appearances, resulting from the different combination of symptoms, is so great, that *Andral* has represented, as different forms, eight different combinations of symptoms which appear in cerebral hyperæmia. We shall only give a short account of the most frequent and important forms.

In many cases of hyperæmia of the brain, disturbances of sensibility, of an irritating character, are the most prominent symptoms. Such patients complain of the head feeling contracted, of more or less severe headache; they are sensitive to bright light and loud noises; have flashes of light before the eyes, and noises in the ears. They go to sleep with difficulty, and the sleep is disturbed by unquiet dreams. In severer cases, there are often dizziness and a feeling of formication in the extremities. The face and conjunctiva are usually reddened, the pulse full and rapid. But we must not consider these symptoms as constant; for, in the most dangerous cases of hyperæmia of the brain, in those induced by excessive mental labor, continued night-watching, etc., the amount of blood in the external organs does not at all correspond to the supply in the brain; and frequently the conjunctiva is not at all injected in such patients, nor is the face flushed; on the contrary, it is pale. Occasionally the above symptoms only last a few minutes. In some patients a few glasses of wine, or some hot coffee, suffice to induce these symptoms of "congestion in the head;" while they remain exempt from them if they keep quiet, and avoid excitement.

The symptoms of hyperæmia of the brain in children, where the motor disturbances are usually greatest, may very much resemble those of meningitis. The two diseases are often confounded, and occasionally they can only be distinguished by their course. Such children have usually suffered from constipation for a few days, have had restless sleep, gnashed their teeth during sleep, or have been frightened out of it by dreams, which disquieted them after waking, and prevented their sleeping again. Then there were vomiting, contraction of the pupil, and twitching of some of the limbs. But these symptoms are often only preliminary to far severer ones, which cause great anxiety. They are followed by general convulsions. In rare cases, these convulsions also occur without premonition, as the first symptoms of great hyperæmia of the brain. The twitching usually begins in one extremity, or one half the face, and rapidly spreads over the body. Occasionally the convulsive movements alternate with tetanic contractions, or certain muscles, particularly those of the back of the neck, are tetanically contracted, while the face twitches, and the extremities are tossed about by clonic spasms. The children do not respond when called, or when the strongest irritants are applied to their skin. They are bathed in perspiration, the abdomen is puffed up, the respiration impaired; the saliva, made frothy by the movements of the masticatory muscles, flows from the mouth. Occasionally there is a pause in the severity of the convulsions, and we hope the attack will pass off; but, after a short remission, the spasms begin again with their former severity, and, in severe cases, the attack may continue, with varying intensity, for many hours. In one child I saw the spasms last over twenty-four hours, with only slight occasional interruptions. Still, in the great majority of cases, the attack ends in half an hour or an hour. In spite of the terror that patients in this state cause the laity and inexperienced physicians, we may give a favorable prognosis, if we can exclude a meningitis. This can usually be done with certainty. Purulent meningitis is, on the whole, a rare disease, which, as we shall hereafter show, only exceptionally occurs in previously healthy children without precedent injury of the head, or disease of the cranial bones. Tuberculous basilar meningitis, a far more frequent disease, often escapes observation till the children have convulsions; but, if we inquire carefully, we shall find that the attack has been preceded by a long illness, and by other symptoms that have been overlooked, or at least undervalued, which we shall hereafter describe. If the child has been well the day before the attack, except some signs of cerebral hyperæmia; if it has had no injury of the head, no otorrhœa, etc., we may be pretty certain that it will be better, if not quite well, the next day. The attack is usually followed by a deep sleep, from which the child

awakens fatigued, but otherwise free from threatening symptoms. In a moderately extensive practice, there is occasion every year to see cases of cerebral hyperæmia in children, which take the above-described very regular course. Not unfrequently several children of the same family will have these attacks at different times, or the same child may have repeated attacks. It is rare for epilepsy to develop from them; but, as we shall hereafter show, repeated hyperæmias may induce hydrocephalus.

In a third form of cerebral hyperæmia the mental symptoms predominate to such a degree that the disease is often mistaken, and, to the great injury of the patient, is sometimes considered as an attack of melancholy; at others, as mania. In the former case, after a few days of headache, disturbance of sensibility, and sleeplessness, the patients are seized with an undefined feeling of anxiety and disquiet. They cannot stay long in one place, go about restlessly, are worried, and are conscience-stricken about slight oversights. There is also delirium, which has the same character as the above-described frame of mind, and results from the attempts to explain it. At first the patients struggle against this delirium, which they occasionally recognize as such, and which they fear, as they think they are "out of their minds;" but they soon weary in this struggle, and give it up. In such cases the sleeplessness is almost absolute; opiates, given by ignorant physicians, have no effect, or, after the exhibition of this remedy, which is injurious and dangerous to the patient, there is a short, restless sleep, from which the patient awakes with all the symptoms increased. In this form of cerebral hyperæmia, which develops chiefly as a result of excessive mental labor, there is usually frequent pulse and other symptoms of fever; but in these very cases a greater amount of blood in the face, etc., does not correspond to the greater amount in the brain; the patients are not high-colored, but are often even pale. From the fever and sleeplessness they rapidly lose strength, emaciate, and, if they do not fall into the right hands, they are in great danger of dying from their disease. Finally, the excitement gives way to apathy, the insomnia to deep sleep, from which the patient cannot be aroused, and in which they die. Far more rarely, there is permanent mental disease.

In other persons, where the psychical disturbance is in excess, it appears in maniacal attacks, with corresponding delirium. This form is especially seen after a long-continued excessive use of spirituous liquors, in that class of topers who for a year will use very little liquor, but when they have begun to drink do not know when to stop. This cannot be easily mistaken for an attack of delirium tremens. The patients are sleepless, run about, fight and bite if they are held, de-

stroy any thing that comes in their way, cry, laugh, or sing. There is also delirium of varying character; usually the patients consider themselves as injured and betrayed, and rage against their enemies and persecutors. The continued muscular exertion throws them into a perspiration, the heart-beat and pulse are accelerated and stronger, the face is usually reddened. This form is also very dangerous if it be mistaken and improperly treated, for then an apoplectic attack, a true apoplexy, or an excessive hyperæmia of the lung with acute œdema, causes death. According to my observation, cases exactly like the following, detailed by *Andral*, are by no means rare; in the Magdeburg hospital, and in the Greifswalder clinic, I have seen several of them within a few years: "For several days a middle-aged man kept up a series of cries sufficient to disturb the whole hospital-ward. These cries ceased suddenly, and on approaching his bed he was found dead. He would not have died more suddenly if struck by lightning. On opening the body, the only lesion found was a lively injection of the cerebral substance."

Lastly, we must mention those forms of cerebral hyperæmia where symptoms of general depression and paralysis appear, and by their sudden occurrence so resemble an apoplexy than an exact diagnosis is often impossible. In some cases the attack is for a time preceded by headache, disturbance of sensibility, dizziness, sleeplessness or psychical disturbances; in others, it occurs without premonitory symptoms. The patient suddenly becomes dizzy; "every thing whirls around with him;" he staggers, all looks dark before him; he loses consciousness and sinks to the ground, either with or without slight spasm. Such an attack, where all activity of the brain is lost, may cause death by the paralysis extending from the brain to the centres of the organic nervous system. More frequently the patients recover consciousness after a time, with an indistinct remembrance of what has passed or without any recollection of it. These apoplectiform attacks, occurring in the course of hyperæmia of the brain, correspond with the above-mentioned epileptiform attacks, as regards the cerebral anæsthesia and the paralysis of the psychical functions, and like them appear to depend partly on obstructed escape of the venous blood, partly on secondary œdema of the brain. As we have previously shown, both states prevent the supply of oxygenated arterial blood, which is indispensable for the excitability of the brain and the entire nervous system. *Traube*, who refers the epileptiform attacks in *Bright's* disease also to œdema of the brain, has advanced the theory that an œdema, limited to the greater hemispheres, induces simple loss of consciousness; when it affects both the hemispheres and medulla oblongata there are loss of consciousness and convulsions. We shall not discuss

the correctness of this hypothesis; it has something seducing about it, but opposed to it is the fact that, if it be true, the same pathological change (compression of the capillaries by serous exudation) has a paralyzing effect on the greater hemispheres and an irritant effect on the medulla oblongata.

Treatment.—Although, in the treatment of hyperæmia of the brain, general and local blood-letting, cold to the head, and derivation to the skin and bowels justly enjoy a good reputation, we must not use one or the other indifferently, or employ all at the same time, in any case that arises. In fact, there is no disease where it is more important to fulfil the causal indications, and to attend to the causes of the disease when prescribing the remedies required by the indications.

In those forms where increased action of the heart and a coincident diminution of resistance in the vessels of the brain have induced fluxion to that organ, the same regimen must be observed as was recommended in the first volume, in the treatment of habitual fluxions to the lungs. If danger threatens, we should bleed; in such cases venesection cannot be replaced by ice-compresses to the head, or by leeching behind the ears.

If there be collateral fluxion to the brain, we must first of all attempt to remove the obstructions to the circulation by which the pressure of blood in the carotids is increased. Evacuating the intestines, by laxatives or by enemata of water and vinegar, often has a marvellous effect, which is induced by nothing else, both in adults who are constipated and suffering from headache, tinnitus, dizziness, etc., and especially in children where constipation is accompanied by convulsions, etc. If these remedies are insufficient and symptoms of depression occur, from which we fear danger, it is proper to draw blood here also, in adults by venesection, in children by leeches to the head.

The treatment must be quite different when the cerebral hyperæmia has resulted from the continued misuse of alcohol or of narcotics, or from excessive mental excitement. In such cases nothing is to be expected from general bleeding, and large venesections are often injurious; on the other hand, the indications are, to apply a bladder of ice to the head, or else moist compresses, that have laid under a tin vessel filled with ice and salt, till they were frozen. In some cases of this variety derivatives are good; by these we attempt to remove the fluxion from the brain by inducing fluxion to the skin, intestines, genitals, or rectum. The most common remedies used with this object are irritant foot-baths, among which those of ice-water, in which the patient only passes a short time, are better than those of hot water, with mustard or salt, and wood-ashes. In acute cases, blisters to the nape of the neck, and in chronic ones the establishment of an issue in the arm—

an old remedy, from which I have seen great benefit—and, lastly, the administration of active purges, are also among the more common remedies. In acute threatening hyperæmia of the brain, croton-oil is peculiarly in repute, while, in chronic cases, pills of aloes, colocynth, jalap, etc., are usually prescribed. If, as occasionally happens, there be fluxion to the brain, instead of to the pelvis, at the menstrual period, we may apply leeches to the cervix of the uterus, or cups to the inside of the thighs. Occasionally, also, the application of leeches about the anus has a wonderful effect, as is shown by the well-known histories of the patients that *Goethe* so much ridicules under the name of "proktophantasmisten."

In congestive hyperæmia resulting from compression of the jugular veins or vena cava, as well as in that occurring from heart and lung diseases, venesection or leeches behind the ears may be employed, if the obstruction to the flow of blood cannot be removed. We have shown that congestion, by arresting the supply of arterial blood to the brain, diminishes or removes the excitability of the brain filaments and ganglia. The greater freedom of escape we give the venous blood, the sooner we shall succeed in removing the symptoms of depression and the paralysis. This may be done by the application of leeches behind the ears, since this moderates the tension in the veins outside of the skull, with which the emissaria Santorini communicate; or by venesection, by which the tension in the anonymous veins is diminished, because less blood enters them from the arm. In such cases we cannot expect any thing from the employment of cold, or from laxatives and blisters.

In hyperæmia of the brain resulting from too much nourishment, instant diminution of the quantity of blood may be urgently indicated, and a well-timed venesection not unfrequently prevents a threatening apoplexy. It is very important to regulate the mode of life of such patients, to show them the danger of prolonged, luxurious meals, and to let them eat little, drink water instead of wine, and walk a great deal.

Of course, in each case, peculiar circumstances will require some deviation from the plans of treatment advised, and different directions as to regimen.

CHAPTER II.

PARTIAL HYPERÆMIA AND PARTIAL ŒDEMA OF THE BRAIN.

A SHORT consideration of partial hyperæmia of the brain will decidedly facilitate the comprehension of the symptoms of diseases of the brain limited to circumscribed spots.

The causes of the partial hyperæmia are to be sought for within the skull. Fluxions and congestions result from extravasations of blood, points of softening and inflammation, tumors, and all other diseases of the brain that affect the circulation at circumscribed spots. If an artery or a great number of capillaries be compressed, or otherwise closed, there is fluxion in the collateral branches; if, on the other hand, a vein be contracted or closed, there is congestion in the capillaries supplying it. Of course, there will usually be fluxion at one place, congestion at another, and anæmia at still others, at the same time. But the circumscribed diseases of the brain not only induce hyperæmia by compression of the vessels, but most of them also excite it by irritation of the surrounding parenchyma. Just as we see hyperæmia and œdema result in the vicinity of tumors, inflammations, extravasations of blood, etc., in all other parts of the body, so they also develop in the brain when it is the seat of these diseases.

On autopsy, it is just as difficult to discover a partial hyperæmia of the brain as it is to make out a general hyperæmia, particularly if the hyperæmia has led to œdema, without the œdema having softened the brain-substance. In some cases, however, in the vicinity of tumors, points of inflammation, etc., we see clearly that the parenchyma is more infiltrated and relaxed, or that there have been small extravasations from the vessels.

The symptoms of partial hyperæmia of the brain are those of irritation and depression, but they are much more limited than the symptoms of general hyperæmia of the brain, and come under the head of so-called local symptoms ("Herdsymptome," *Griesinger*). Among these are circumscribed headache, glimmering or sparks before one eye, or blindness of one eye, contraction or dilatation of one pupil, noise or deafness in one ear, neuralgia or anæsthesia limited to one nerve, but especially spasms, contractions, or paralysis, affecting only one-half of the body, one extremity, or a single group of muscles, and, lastly, partial disturbance of the mind.

The grade and extent of the partial hyperæmia of the brain vary with the greater or less amount of blood contained in the organ, and with the phases and stages of development of the point of disease that they surround. This explains why the local symptoms depending on partial hyperæmia of the brain are sometimes more prominent than at others, or may even disappear and return again. Since, in all severe structural disease of the brain there is complete loss of function of the affected part, whether the trouble there be the development of a tumor, or that the nerve-filaments and ganglion-cells have been broken down or destroyed by an extravasation of blood, the only symptoms that we can consider as immediately due to severe local disease of the brain

are partial anæsthesia, partial paralysis, and the loss of certain mental functions.

If, in the course of an apoplexy, of an abscess of the brain, or of a cerebral tumor, etc., we have symptoms of partial irritation, as well as those of partial paralysis, the former cannot possibly depend on the disease itself, but must be due to the anomalies of circulation in its vicinity. Moreover, all temporary symptoms of paralysis occurring in the course of local destructive diseases do not depend on the disease itself, but mostly on the disturbances of circulation around it. (When speaking of diseases encroaching on the space in the skull, we shall show that temporary paralysis may also result in other ways.) The fact that in apoplexies, tumors, abscesses, etc., where complete or even partial *restitutio ad integrum* is not conceivable, paralysis not unfrequently decreases or entirely disappears, seems at first difficult to explain, but the explanation is simple when we bear in mind that the symptoms of paralysis may depend on a collateral œdema in the vicinity of the disease, and that the extent of this œdema varies greatly.

Lastly, the most varied diseases of the brain would induce the same symptoms, if they had similar locations and extent, if the disturbances of circulation in the vicinity of the different diseases did not vary. But the disturbances of circulation in the vicinity of a tumor are different from those in the vicinity of an abscess, and these again differ from those about a portion of brain broken down by an extravasation of blood. This, to some degree, explains the difference of symptoms in different diseases of the brain that have the same seat and extent.

The treatment of partial hyperæmia is to be conducted on the same plan as that for general hyperæmia of the brain. If we lessen the supply of blood, or facilitate its escape, we moderate the local fluxion or congestion.

CHAPTER III.

ANÆMIA OF THE BRAIN AND ITS MEMBRANES.

Etiology.—For a time the possibility of anæmia of the brain was denied, just as that of hyperæmia was, and on the same grounds which were given in the first chapter. We have already called attention to the errors in these conclusions, and shall only mention here that, independently of the numerous autopsies where the brain has been found anæmic, *Donders*, *Küssmaul* and *Tenner* have observed excessive anæmia through an opening (covered by a glass plate) in the skull of living animals.

The causes of anæmia of the brain are—

1. Those that diminish the entire amount of blood in the organ. Among these belong not only abstractions of blood and spontaneous hæmorrhages, but extensive losses of fluid, considerable exudations, and tedious, particularly feverish, diseases. Unfortunately, it occasionally happens that, in internal hæmorrhage, anæmia of the brain is mistaken for hyperæmia, and treated accordingly. The form of the disease which, since the time of *Marshall Hall*, has been known as hydrocephaloid, is particularly common in children who have suffered from continued diarrhœa. Typical examples of this are not unfrequently seen as a result of extensive hepatization in weak persons with pneumonia. But protracted fevers also consume the flesh and blood of the patient, induce general poverty of the blood, and, as one symptom of it, anæmia of the brain. In all of these diseases, blood and the fluids of the body are lost or used up too rapidly; on the other hand, the amount of blood may be diminished by its formation being limited from insufficient supply of nourishment. Thus, in persons who have died of starvation, the most marked symptoms of anæmia of the brain have been observed before death (as *Gerstenberg* has described in his "Ugolino," in very vivid terms, it is true, but still quite accurately).

2. This affection not unfrequently results from the overloading of other organs with blood. The best example of this form are the cases where it is induced by the application of *Junod's* cupping-boot, by the injudicious use of which the anæmia may readily become dangerous. This also explains why, when the heart's action is weak, a person faints more readily when standing up than when lying down. It is evident that, in the upright position, the lower extremities will become overloaded with blood, if the propelling power is insufficient to overcome the obstruction caused by the weight of the venous blood in this long course. On the other hand, in diminished action of the heart, the obstruction from the weight of blood in the short carotid artery must have a very inferior influence on the occurrence of anæmia of the brain in the upright position.

3. Another cause is compression or obstruction of the arteries supplying the brain. In almost all of the cases of this class that have been reported, the obstruction was artificially caused by ligation of the carotid. In a few cases only the carotid or vertebral arteries were compressed by tumors or closed by emboli.

4. Cases where, from mental excitement, without lessening of the heart's action, there are paleness of the cheeks and even loss of consciousness and other symptoms of insufficient supply of blood to the brain, seem to indicate that anæmia of the brain may also be caused by abnormal innervation or spasmodic contraction of the arteries.

5. Anæmia of the brain is the necessary result of diminution of the space in the skull by exudations, extravasations, or tumors of the brain and its membranes. Under this form would come a variety which does not belong in our domain, that is, the cases of anæmia which necessarily result from depressed fractures of the skull. We lay peculiar stress on the anæmia of the brain resulting from encroachment on the cranial cavity, which is the most frequent form of the disease, because we believe that the so-called symptoms of pressure in apoplexy, tumors, the various forms of hydrocephalus, and other diseases encroaching on the intercranial space, are not immediately referable to the pressure on the brain-substance, but to the consequent anæmia. Other observers also, among them *Traube* and *Leyden*, have recently come to my conclusion.

Since it is not merely the presence of blood in the vessels of the brain, but the supply of oxygenated arterial blood that is indispensable for the normal functions of the organ, it is evident that, even where the absolute amount of blood in the brain is not diminished, but where its circulation and distribution are changed so that only a small amount of blood enters through the arteries, and but little escapes through the veins, the same symptoms must arise as in true anæmia. And the experiments of *Küssmaul* and *Tenner* entirely confirm the experience of pathologists, that, in degeneration of the heart from non-compensated valvular obstruction, and in other diseases impairing its action, there is an overloading of the veins at the expense of the arteries, and a retardation of the circulation, that is, the same symptoms that occur in anæmia of the brain.

Lastly, we must mention that, without a diminution of the amount of blood in the brain, and where that fluid is normally distributed in the arteries and veins, symptoms very similar to those of anæmia may result from the blood being too poor in red corpuscles. This symptom also is readily explained, for we are fully justified in considering the red corpuscles as the "bearers of oxygen." Now, if this be so, a diminution of red corpuscles will affect the supply of oxygen to the brain, just as a diminution of the blood would.

Anatomical Appearances.—The substance of the brain is discolored; the gray substance appears paler and more resembles the white. The latter is very milky and shining. On section, few if any blood-points are seen on the cut surface. The vessels of the cerebral membranes are empty and collapsed. We do not always find a considerable amount of fluid in the subarachnoid space. *Küssmaul* and *Tenner* could not prove, on examination, any increase in the cerebro-spinal fluid which, on theoretical grounds, they had expected to find.

Symptoms and Course.—The symptoms of anæmia of the brain

that comes on suddenly, and quickly attains a high grade, differ from those due to one which comes on slowly and is less severe. In the former case the patients become dizzy; every thing appears dark before them; they become insensible to impressions and incapable of movement; their pupils dilate, their respiration becomes slow, and they lose consciousness; they sink to the ground, usually with slight spasms. In most cases the patients come out of this fainting-fit in a short time; in other cases, usually termed apoplexia nervosa, consciousness does not return, the swoon ends in death. Anæmia of the brain artificially induced in animals by free bleeding, or by ligation of all the arteries supplying the brain, has just the same symptoms; only the convulsions are usually more severe and more prominent than in persons whose brain has suddenly become anæmic. The symptoms of paralysis in sudden, extensive anæmia of the brain are more easily explained than are the convulsions. The former unmistakably depend on the arrested supply of oxygen to the brain. It is known that ligation of the abdominal aorta immediately induces paralysis of the lower half of the body, whose nerves are thus deprived of arterial blood. But how shall we explain the convulsions? *Henle* thinks that in anæmia of the brain the blood from the venous plexuses of the spinal marrow and the cerebro-spinal fluid from the spinal canal press toward the brain, and that the medulla oblongata and parts at the base of the brain are thus thrown into a state of excitement. But *Küssmaul* and *Tenner*, after ligating the afferent vessels, found not only the greater hemispheres, but also the medulla oblongata, bloodless; hence the convulsions cannot be referred to congestion of the medulla. But it is just as remarkable, and as contrary to all experience, to suppose that there should be increased excitement of the nerve-filaments and ganglion-cells, with consequent convulsions, from absolute anæmia. *Küssmaul* and *Tenner* distinctly state that, on autopsy of animals whose cerebral arteries had been ligated, the arteries at the base of the brain contained a "slight amount of blood," while all others were found "entirely empty;" this observation gives a small point on which to hang an explanation. For we might suppose that the ligation of those vessels caused absolute anæmia in the greater hemispheres and their consequent paralysis, but (from the anastomosis of the cerebral and spinal arteries) in the parts at the base of the brain it only induced oligæmia and consequent morbid excitement.

In anæmia of the brain that comes on slowly, just as in hyperæmia, at first there are usually symptoms of irritation, subsequently those of paralysis. To explain this correspondence, the hypothesis has been advanced that a certain tension of the molecules of the brain is necessary for its normal activity and that an increase or diminution of this

tension, by too great or too slight a fulness of the vessels, modifies the excitability of the brain in the same way. I have already said that this is a hypothesis, and, I may add, that it is difficult for me to believe that, in anæmia of the brain, the symptoms of irritation depend on an inconsiderable, and those of paralysis on a decided, diminution of the normal pressure of the blood-vessels on the brain. On the other hand, it is a physiological fact that the excitability of a nerve is increased a short time before it is entirely lost, and that the greatly-increased excitability of a nerve is not a sign of increased normal nutrition, but, on the contrary, of its diminution. It is true we do not know why this is, but the knowledge that it is so renders it less strange that, in gradually-developing anæmia of the brain, symptoms of irritation should, as a rule, precede those of paralysis, and that, where the anæmia does not attain a high grade, only the symptoms of irritation should be seen.

Occasionally the symptoms of anæmia consist chiefly or exclusively of disturbances of *sensibility*. The patients complain of severe headache, either in the forehead or occiput; they are sensitive to light and sound, have flashes before the eyes, noises in the ears, dizziness, etc. These symptoms occur most typically after severe metrorrhagia and other extensive losses of blood, and frequently only the consideration of the cause, the pulse, color of the skin and lips, and the symptoms of want of blood in other organs, enable us to decide that there is anæmia, and not hyperæmia, of the brain.

In other cases of anæmia of the brain, particularly in children, the *motor* disturbances are more prominent. The symptoms of anæmia of the brain in children from exhausting diarrhœa and other debilitating causes, so-called *hydrocephaloid*, often so closely resemble those of acute hydrocephalus, that the distinction of the two states may be very difficult. *Marshall Hall* divides hydrocephaloid into two stages, one of irritation, one of torpor. In the first stage the children are very restless and capricious, constantly toss about in bed; readily frightened, they cry out in their sleep, gnash their teeth; the face is usually flushed, the pulse frequent, and temperature elevated. There is al most always slight twitching of some limbs, frequently also there are general convulsions. In the second stage the children collapse, become entirely apathetic, no longer attend to objects held before them; the eyelids are half closed, the pupils insensible to light; respiration becomes irregular and rattling; finally death occurs with symptoms of coma. Since we regard the so-called symptoms of pressure in diseases of the brain and its membranes (among which are effusions into the ventricles), which encroach on the cranial cavity, as due to compression of the capillaries and obstruction of the supply of arterial blood

to the nerve-elements of the brain, we do not consider the great similarity of the symptoms of hydrocephalus to those of hydrocephaloid as very strange. Different as are the modes of origin of the two diseases, we believe that in both of them there is ultimately the same pathological disturbance, that is, capillary anæmia.

Lastly, in anæmia of the brain, morbid symptoms in the *mental* functions preponderate; there are sleeplessness, great excitement, delirium, etc. In some cases this state increases to paroxysms of frenzy and decided maniacal attacks. The latter are seen in persons who have had no food or drink for a long time, but they are also not unfrequently seen in weakly, bloodless patients, if their anæmia has been greatly increased by exhausting diseases and abstractions of blood.

Treatment.—If the anæmia of the brain be one symptom of general impoverishment of the blood, this is to be treated by limiting the consumption and increasing the supply of suitable nourishment. A consideration of the etiology and other circumstances of each case gives the indications for treatment. If profound and continued faintness, convulsions, and other signs of excessive hyperæmia of the brain come on after extensive loss of blood, even transfusion may be necessary. In treating the exhausting diarrhœa of infants, we should think early of the danger of hydrocephaloid, and attempt to prevent it by giving raw flesh, wine, etc. If, nevertheless, the symptoms above described come on, a false interpretation of them is very dangerous. If the practitioner be misled into applying leeches or employing debilitating treatment, the children usually die. But if he sees through the condition and, in spite of the restlessness, twitching, and consequent sopor, gives concentrated broths and large doses of stimulants, camphor, ether, but especially of strong wine, he often attains the happiest and most surprising results. In the form of anæmia of the brain which occurs as one symptom of general impoverishment of the blood, it is also very important that, until the normal quantity and quality of the blood is reëstablished, we see that the heart does not receive too little blood; and it is just as important for us to combat any temporary weakness of the heart, that prevents it driving its blood energetically into the arteries. Very many patients, with impoverished blood, and convalescents die, solely because the physician has neglected to give them the strictest orders to maintain a horizontal position. If we permit exhausted patients to rise to stool, or to leave the bed too soon, the feet are readily overloaded with blood, the heart receives too little; consequently a sufficient supply does not go to the brain; the patient swoons, and not unfrequently does not awake from the fainting-fit. On this very point I have had an experience in my private practice which will always prove a warning to me, not to permit convales-

cents to leave their beds too early. To prevent the second danger from temporary lessening of the heart's action in anæmic patients, besides the horizontal position, we may order irritating remedies both by inhalation and internally. The patients should not use these remedies constantly, but only occasionally when they feel an attack threatening. Indeed, it may be important to have Cologne-water or Hoffman's anodyne instantly at hand on such an occasion. Trephining is occasionally the only remedy for anæmia caused by encroachment on the cranial cavity; however, in recent times this is very properly limited to those contractions of the space caused by depression of the cranial bones. Paradoxical as it may seem on superficial observation, we must, nevertheless, say that a venesection often has the best effect on the course of anæmia of the brain due to a contraction of the intracranial cavity. We shall give the reasons for this assertion more fully when speaking of apoplexy, and shall only say here, that a venesection hastens the escape of venous blood from the brain, and thus facilitates the entrance of arterial blood.

CHAPTER IV.

PARTIAL ANÆMIA AND PARTIAL NECROSIS OF THE BRAIN, THROMBOSIS AND EMBOLISM OF THE CEREBRAL ARTERIES—SOFTENING.

Etiology.—Partial anæmia of the brain occurs—1. When the access of blood to certain sections of that organ is prevented by closure of the afferent vessels. 2. When collateral œdema develops in the vicinity of apoplexies, of points of inflammation, and softening, and of tumors, etc. 3. When the capillaries of certain sections of the brain are compressed by extravasation of blood, by tumors, or other diseases contracting the cranial cavity.

In regard to the pathogeny of the first form of partial anæmia of the brain, which develops in the parts supplied by obstructed arteries, we must call attention to the following points:

In rabbits, the symptoms of anæmia of the brain do not occur till both carotids and both vertebral arteries have been ligated. If all these vessels be not ligated, those that have been left free supply plenty of blood, and the free anastomosis of the cerebral arteries among themselves prevents anæmia even of those parts which draw their blood chiefly from the vessels ligated. In the human being it is somewhat different; here, after ligation of one carotid, there is occasionally a symptom which certainly depends on anæmia of one side of the brain, that is, paralysis of the half of the body on the opposite side. In other cases of ligation of the carotid in man, this symptom does not

arise, which proves that in these cases the endangered hemisphere receives a sufficient amount of blood from collateral branches, particularly through the circle of *Willis*. The reason for this difference is not fully known. *Hasse* thinks that in the former case the continuation of the thrombus from the point of ligation beyond the circle of *Willis* prevents the formation of a collateral circulation. On closure of the internal carotid or of the vertebral artery on one or both sides, partial anæmia does not generally occur, since a collateral circulation is usually quickly established through the circle of *Willis*. On the other hand, closure of an artery originating above the circle of *Willis*, e. g., the arteria fossæ Sylvii, almost always induces anæmia of the part supplied by the obstructed vessel, since here circumstances are much more unfavorable for the formation of a collateral circulation.

The pathological processes that chiefly induce closure of the arteries of the brain are in some cases compression by tumors, but in most cases obstruction by thrombosis formed at the spot or by emboli from some other part.

It is only exceptionally that the blood coagulates in cerebral arteries whose walls are healthy (marasmic thrombosis). As a rule, the thromboses form at points where, as a result of chronic endarteritis, or, as is usually said, of atheromatous degeneration, the calibre of one of the vessels of the brain is diminished and its inner wall roughened.

The emboli by which cerebral vessels are closed are almost always detached clots of fibrin, that have been deposited, in endocarditis or valvular disease, on rough parts of the valves, or else portions of the valves themselves, that have been washed off by the current of the blood. These rarely come from necrotic points in the lungs, or from thromboses of the pulmonary vein. In one very instructive case, published by *Esmarch*, an embolus obstructing the internal carotid came from an aneurism of the carotid, having been set free by the manipulations during an examination.

We have but little to add in regard to the etiology of this form of anæmia of the brain. Since the atheromatous affection, which most frequently causes thrombosis of the cerebral vessels, generally occurs in advanced age, the stoppage of the arteries of the brain by thromboses is almost exclusively seen in aged persons, and plays an important part in the diseases of old age. The case is different with embolism of the cerebral arteries; this is seen in young persons also, since endocarditis and valvular disease of the heart occur at all ages.

The pathogeny of the partial necrosis, which results in many cases of thrombosis and embolism of the cerebral arteries, is perfectly evident. This form of softening of the brain is analogous to the gangrene of the extremities induced by closure of the vessels. In both cases

the death of the tissue is due to abstraction of the supply of nutritive material; but the necrosed parts within the skull, not being exposed to the action of the atmosphere, are rarely decomposed. This only happens when the embolus, that has stopped the vessel, comes from a suppurating spot, and transfers with it a tendency to suppuration. Closure of a vessel induces necrosis more readily the later and more incompletely a collateral circulation is established. If the degeneration of the walls of the vessels that has induced a thrombosis of the cerebral arteries be extensive, the collateral branches, the elasticity of whose walls is diminished, cannot dilate sufficiently to supply the place of any large arteries that may be closed, hence the partial anæmia is only partly removed, and the anæmic part softens. Whether or not closure of a cerebral artery by an embolus shall induce necrotic softening depends chiefly on the seat of the obstruction, since in such cases the walls of the vessels are usually healthy and distensible. If, as rarely happens, there be anæmia in the parts chiefly supplied by a vessel which has been obstructed by an embolus before reaching the circle of *Willis*, it will generally pass off soon, and no necrosis results; if, on the other hand, a vessel be closed by an embolus beyond the circle of *Willis*, necrosis is the usual termination of the partial anæmia.

The second form of partial anæmia of the brain, due to development of collateral œdema around an apoplexy, or a point of inflammation or softening, or a tumor, etc., has already been mentioned as the not unfrequent termination of excessive fluxionary hyperæmia. When speaking of the different diseases of the brain, we shall frequently recur to this form.

The third division that we have made of partial hyperæmia of the brain, resulting from compression of the capillaries in certain sections of the brain by extravasations of blood, tumors, and other diseases contracting the space in the skull, has hitherto been too little appreciated. Along with the best authorities, I myself denied the occurrence of "partial cerebral pressure." From the fact that the brain is incompressible and is enclosed by an inelastic capsule, I reasoned that an increased pressure, acting on any part of the brain, would spread evenly over the entire organ. In support of this view, I advanced the popular illustration that, if a cork be driven forcibly into a bottle, the latter is not by any means always broken at the neck, but just as often at some distance from the point where the force was applied, perhaps at some particularly weak part. However, a series of observations where there was no doubt that those portions of the brain where the disease was located were far more bloodless than the rest of the brain, and a careful consideration of all circumstances af-

fecting the space in the skull, have shown me that, in the above reasoning, I overlooked an important fact and hence came to a false conclusion. Indeed, in spite of the brain being incompressible, and being surrounded by an unyielding capsule, partial pressure on it may occur; this is so, because the cranial cavity is divided into three chambers by two tensely-stretched membranes, because the falx and tentorium protect, to some extent at least, parts of the brain lying on one side of them from pressure acting on the other side.

The three chambers of the skull communicate with one another, it is true, and, if the cerebral substance were liquid, pressure on any part of it would affect the whole organ equally, in spite of the tense septa traversing it. But the consistence and tenacity of the cerebral substance, which only permit a slight protrusion of a section of brain pressed from an opposite chamber of the skull toward the excavation of the tentorium or the lower border of the falx, cause portions of the brain lying in one chamber to be protected to some extent from pressure on those lying in one of the other chambers, in spite of the openings in the septa, particularly the large opening in the falx. The protection afforded by the tentorium is greater than that given by the falx, and the posterior lobes of the cerebral hemispheres are far better protected against a pressure acting on the opposite hemisphere than the frontal lobes are, because the falx is much broader posteriorly, and hangs much farther down than it does anteriorly. In subsequent chapters we shall frequently refer to these circumstances also, whose great significance in the explanation of the symptoms of disease encroaching on the cranial cavity I was, to the best of my knowledge, the first to point out.

Anatomical Appearances.—Partial anæmia of the brain cannot by any means always be definitely made out in the dead body. The distribution of blood after death and during life is not the same; places which, during life, were distinguished by their vascularity from other less vascular parts, often become just as bloodless as the latter after death. In the cutis, where we have an opportunity for comparison, we may see this daily; in the brain the state of affairs is just the same. It is remarkable that emboli are almost always found in the left arteria fossæ Sylvii. Perhaps this is partly because the left carotid artery leaves the arch of the aorta almost in the direction of the current of blood, while the innominate forms a considerable angle with it (*Rühle*).

The necrosis resulting from obstruction of the vessels and insufficient development of collateral circulation induces relaxation and softening of the brain-substance; hence necrosis caused by anæmia is designated as a peculiar form of softening of the brain, as "simple" or "yellow" softening. Corresponding to the most frequent and obsti-

nate obstructions of the vessels, the softening is usually in the greater hemispheres and chiefly in their medullary substance. It varies in size from that of a bean to a hen's egg. The grade of the softening differs. In the highest grades the cerebral substance, at the point of necrosis, is found changed to a moist, gelatinous trembling pulp. The color of the softened point is sometimes white or grayish-white, sometimes more yellowish. In the former case there is usually a reddish tint about the periphery, due partly to dilatation, partly to rupture of the capillaries and escape of blood from them. In the first of the three forms of partial anæmia which we have described, that due to thrombosis or embolism of a cerebral artery, on autopsy we do not find the parts supplied by the plugged artery remarkably pale, but they are not unfrequently studded with small capillary hæmorrhages, particularly at the periphery. This exactly corresponds with what is found in other organs when there is stoppage of the vessels by thromses or emboli; but, as we said when speaking of hæmorrhagic infarctions of the lung, it is difficult to explain. On account of the difficulty of deciding whether a portion of brain has been anæmic during life, we should make it a rule to seek most carefully for any obstruction of the cerebral arteries, particularly of the arteria fossæ Sylvii, in any cases where the patient has died from a chronic brain disease or from an acute one beginning with a sudden occurrence of hemiplegia, unless the autopsy gives some other satisfactory explanation of the symptoms. Before attention was called to the occurrence of this anomaly, autopsy, in cases of severe brain-disease with hemiplegia, often furnished nothing satisfactory. There was nothing left but to suspect an "intravascular apoplexy," which did not at all explain the occurrence of the hemiplegia. The yellow color of the affected part often depends on this capillary hæmorrhage, and is due to the infiltration of the disintegrated brain-substance with escaped and altered coloring matter of the blood. After the disease has existed some time, we find the affected part changed to a cellular network filled with a chalky, milky fluid (*Durand-Fardel's* infiltration celluleuse). On microscopical examination of necrosed portions of brain, we usually find only remains of nerve-filaments, granular cells, which correspond to the ganglion-cells or neuroglia-nuclei that have undergone fatty degeneration, coloring matter, and masses of detritus.

Partial anæmia of the brain caused by collateral œdema, occurring in the vicinity of circumscribed disease, can occasionally be recognized on autopsy by the affected part having a peculiar moist lustre and diminished resistance, as well as by its becoming very slightly prominent on a section through the brain. When the disease is more severe, the brain-substance is still more relaxed, and finally a state may

occur which is usually designated as white (hydrocephalic) softening. Small capillary extravasations are often found in the vicinity of tumors, abscesses, or along with collateral œdema.

The form of partial anæmia of the brain, induced by diseases encroaching on the cranial cavity, is the variety most readily recognized on autopsy. The pressure exercised by large extravasations of blood and extensive tumors is so great that, not only the capillaries and finer arteries and veins of the brain-substance, but also the larger vessels of the meninges, that are subjected to this pressure, are compressed and bloodless. If the disease be in one of the large hemispheres, this becomes more prominent after the skull is opened, so that on the affected side the dura mater appears more tense than on the other side. If the dura mater be opened and turned back, we see that the surface of the diseased hemisphere is remarkably even, that there is very little, if any, liquid in the subarachnoid space, that the convolutions are lower, the furrows shallower, and that the vessels of the pia mater are not so full or are quite empty. Lastly, also, on section through the brain, we cannot fail to see a decided difference in the two hemispheres in regard to their color and as to the number of blood-points appearing on the cut surface. In those cases where the falx and tentorium have to a certain extent given way to the pressure propagated to them, the falx showing a convexity toward the healthy side, the tentorium being flattened, or, when the disease is in the posterior cranial fossa, being more strongly curved, it is certain that the capillaries are compressed in those portions of the brain where the disease encroaching on the space is located. But at the same time it is found that the anæmia does not remain limited to the part first affected, but extends to other parts subsequently, although to a less extent.

Symptoms and Course.—Anæmia limited to one portion of the brain induces the so-called "herdsymptome" (page 194); if the anæmia be absolute, these consist of symptoms due to complete loss of excitability in the anæmic portions of the brain; if it be not absolute, there may even be signs of increased excitability, or morbid excitement of the affected portion of the brain. In the vicinity of the anæmia, often even through the entire brain, the circulation is disturbed, so that, besides the direct symptoms of partial anæmia, we may have those of more or less extensive secondary disturbances of circulation. However, neither the "herdsymptome," nor the symptoms of secondary disturbance of the circulation in the brain, nor a combination of the two, are pathognomonic of partial anæmia of the brain; on the contrary, we must distinctly state that each of these also occurs in many other diseases of the brain. Not unfrequently, we can recognize one or other form of partial anæmia of the brain, and exclude

other local diseases there, by basing our diagnosis on the etiological conditions, the sequence of the symptoms, the existence of symptoms corresponding to the peculiar frequence of certain forms of disease in certain sections of the brain, as well as on the course of the disease.

We shall first describe the symptoms of the occurrence and course of partial anæmia of the brain due to obstruction of the vessels, and show how it is often possible to diagnose this form of partial anæmia with probability or with certainty by attending to the inducing causes. However, the etiology of thrombosis of the cerebral vessels differs from that of embolism, and the other factors also, which might be of aid in diagnosis, are not exactly the same in thrombosis and embolism of the cerebral arteries; hence we shall speak of them separately.

Since thrombosis of the cerebral vessels most frequently depends ultimately on atheromatous degeneration of the vascular walls, and as this occurs chiefly in old age, we are more apt to suspect thrombosis, and consequent softening of the brain, in an old, decrepit person, who has the symptoms of severe brain-trouble, than in a young, vigorous one, having the same symptoms. If the peripheral arteries be rigid and tortuous, there is still greater presumption that the arteries of the brain are also degenerated, and that the brain-symptoms are due to this degeneration. However, the condition of the peripheral arteries does not furnish any certain proof of that of the cerebral arteries. In many cases the degeneration is confined to the latter; in other, rarer cases, while the peripheral arteries are extensively degenerated, the cerebral arteries remain free. Moreover, partial anæmia of the brain, and softening of the brain due to necrosis of the anæmic portion, is not the only brain-disease caused by atheromatous degeneration of the cerebral arteries. Experience shows that atheromatous degeneration of the walls of the vessels usually induces dilatation of the larger arterial trunks, and, on the contrary, contraction of the smaller arteries. The atheromatous arteries of the brain are also usually contracted for a long while before they are closed by thromboses. Hence the symptoms of thrombosis, or, rather, of the partial anæmia and partial necrosis of the brain depending on it, are almost always preceded by premonitory symptoms, either of disturbance of circulation, induced by the contraction of certain cerebral vessels, or by symptoms of senile cerebral atrophy, which has been caused and hastened by degeneration of the cerebral vessels. The patients complain of pain in the head, dizziness, ringing of the ears, flashes before the eyes, loss of memory and power of thought; they are apathetic and indifferent, and much inclined to sleep, but their sleep is disturbed by uneasy dreams. As a rule, very small arteries are at first obstructed, either by the atheromatous process causing their entire obliteration, or be-

cause their calibre is contracted, and then obstructed by a thrombosis. The anæmia resulting from closure of these small vessels is limited in extent, and hence may readily be removed by an increased supply of blood through neighboring vessels. We must bear this in mind when, in a marasmic patient, who for some time has had the brain-symptoms that we designated as premonitory, limited regional symptoms (herdsymptome) occur, and, after lasting for a time, disappear again. Among these symptoms are inability to say different words, loss of memory for certain names and numbers, pain, or a feeling of formication, or of certain limbs going to sleep, occasionally only of certain fingers or toes, contractions and paralysis, which are also occasionally limited to certain fingers or toes, etc.

Many authorities have explained this variation of symptoms, particularly the occurrence and disappearance of paralysis, as a peculiar symptom of softening of the brain. This is a false view of the matter. In cases where the symptoms have presented this variation, if softening of the brain be found on autopsy, the softening did not occur at the time the symptoms changed, but at a later period, when they were constant. On the other hand, the occurrence and disappearance of circumscribed paralyses are certainly, to some extent, characteristic of partial anæmia of the brain resulting from atheromatous degeneration and thrombosis of small cerebral arteries, and are rapidly removed again by the establishment of collateral circulation. (The occurrence and disappearance of circumscribed paralyses do not render it certain that there is thrombosis of small arteries of the brain; the same symptom is also seen from small extravasations. See Chapter V.) If a large artery, or several small ones going to the same part of the brain, be closed by thrombosis, as we have already shown, a collateral circulation cannot be established, particularly if the degeneration of the walls of the vessels be extensive, and then the affected portion of brain loses its functional power forever. There are some parts of the brain—for instance, the large medullary masses of the hemispheres—that may be destroyed without any apparent loss of function. This fact, which is proved by numerous examples, explains the occurrence of those cases of softening during whose course there have never been any symptoms of paralysis. We must know this in order to understand that it is occasionally quite impossible to diagnose softening of the brain, and to distinguish it from simple senile atrophy, because the most important point for the differential diagnosis is wanting. But, far more frequently, the results of thrombosis of a large artery, or of numerous small ones, extend to parts of the brain, whose loss of function induces paralysis, and even hemiplegia, particularly to the corpus striatum and thalamus. The arteries supplying the great hemi-

spheres, and the above-mentioned large ganglia lying in them, with blood, are the ones that are most frequently closed by thromboses; and, even if the anæmia directly resulting from the obstruction, and the consequent softening, do not extend to the corpus striatum and optic thalamus, these parts will readily lose their functional power from the collateral œdema about the point of softening, or from the capillaries of the entire hemisphere being compressed by it. If a large vessel be closed by a thrombosis originating from the walls, and growing slowly, or if numerous smaller arteries be closed one after the other, the paralysis comes on slowly, and increases gradually. Cases running this course are the most readily recognized; for, although gradually-forming and slowly-progressing paralysis also occurs in many other cerebral diseases, if this symptom arise in an old marasmic patient, who has had the previously-described symptoms, we must first think of thrombosis of the cerebral vessels, and of the form of softening of the brain at present under consideration. While the symptoms of paralysis, which are very often and unaccountably accompanied by contractions of the paralyzed part, gradually increase and extend, the patients become more apathetic, grow imbecile, pass their excrements involuntarily, have bed-sores, and finally die of marasmus and coma. The course is different when a large vessel or numerous small ones are rapidly closed by thrombosis. In such cases hemiplegia occurs suddenly, and the symptoms may be very similar to or identical with those of cerebral hæmorrhage. This correspondence is easily explained. In cerebral hæmorrhages, also, most frequently, the corpus striatum and thalamus are either broken up, or their functional activity is arrested by the compression that capillaries of the whole hemisphere suffer from large effusions. In cerebral hæmorrhages also, hemiplegia usually occurs suddenly. Moreover, ruptures of the cerebral arteries usually occur in old persons, and are most frequently due to the same disease of the vascular walls that generally induces thrombosis. We shall not enumerate the symptoms given for a differential diagnosis, and shall only refer to an assertion of *Bamberger's*, for which science is indebted to such an undisputed authority on diagnosis. *Bamberger* says that, in his notes, he finds seven cases where there was an error of diagnosis, and the real state of affairs was only discovered at the autopsy; he considers it impossible to avoid this error, and says he seldom ventures to make an absolute diagnosis of cerebral hæmorrhage from an apoplectic attack.

Partial anæmia and necrosis of the brain due to *embolism* are also almost always preceded by characteristic premonitory symptoms. But these are not brain-symptoms, as they were in the previous form of anæmia of the brain; they are those of the diseases which

almost exclusively cause embolism of the cerebral arteries, that is, of valvular disease of the heart, of endocarditis, or of severe destructive disease of the lungs. The occurrence of these premonitory symptoms, and the presence or absence of valvular disease, endocarditis, or severe disease of the lung, have such an effect on the diagnosis between embolism of a cerebral artery and other brain-diseases, that with the same set of symptoms we may diagnose embolism if we find them, and exclude it with certainty if they are absent. The sudden shutting off of arterial blood from the part of the brain supplied by the obstructed artery, instantly arrests its functional power. Experience shows that emboli almost always lodge in the arteria fossæ Sylvii, particularly the left one; as the closure of this large artery causes great anæmia of the parts supplied by it, we may readily see that sudden hemiplegia, especially of the right side, is the most important symptom from which we can diagnose embolus in the cerebral arteries, if it occur in a patient with valvular disease, etc. The entire loss of consciousness, the apoplectic attack, which usually accompanies the commencement of hemiplegia, when the arteria fossæ Sylvii is stopped by an embolus, is more difficult to explain. I think that this symptom is most probably due to the diseased hemisphere being decidedly swollen by collateral œdema, and that, as occurs in large extravasations of blood, the opposite hemisphere is not sufficiently protected from the pressure by the falx, which only offers a limited amount of resistance. In embolism of peripheral arteries at least, I have always found a very decided and wide-spread œdema in the vicinity of the obstructed vessel, and have witnessed considerable enlargement of the spleen from embolism of the splenic artery. It is evident that a hemiplegia occurring suddenly with an apoplectic attack may readily be mistaken for a cerebral hæmorrhage. In some cases, it is true, the age of the patient gives grounds for distinguishing a hæmorrhage from an embolism. Hæmorrhages occur chiefly, although not exclusively, in advanced age, embolism comes in persons of any age; hence, in young persons, the presumption is in favor of embolism. However, the only way of avoiding error is the careful examination of the heart and lungs. The certainty that the diagnosis receives from the discovery of valvular disease is still more increased if we can also find a coincident embolism of a peripheral artery or of one of the internal organs, such as the spleen or kidney. In most cases death occurs sooner or later after the attack, with the symptoms of general paralysis; in other cases consciousness returns after a time. The symptoms of paralysis rarely disappear; this is sufficiently explained, as we have previously shown, by the difficult establishment of a collateral circulation.

Partial anæmia of the brain, due to collateral œdema in the vicinity

of abscesses, tumors, and other local diseases, induces symptoms of irritation or paralysis, according to the degree of the anæmia; these symptoms complicate those directly depending on the original disease, and are due to functional disturbance of portions of the brain lying beyond the actual disease. On autopsy, it is usually difficult or even entirely impossible to decide, whether œdema and capillary anæmia exist in the vicinity of an abscess, tumor, etc., and how far they extend. But we have a right to suppose that the vicinity of these points of disease is in a similar state to that of parts affected in the same way, which are exposed to observation; and we are the more justified in this supposition, because for a long time it has not escaped the more accurate observers that, in many cases of partial disease of the brain, symptoms occur that cannot depend on the coarser structural changes of the brain found on autopsy, but must be referred to an imperceptible participation of other portions of the brain lying in the vicinity of the affected part. It is most probable that this participation depends on disturbance of the capillary circulation and the occurrence of œdema, because in other parts of the body also these anomalies often leave no traces. Occasionally the symptoms observed during life give a better means of judging of the extent of the secondary disturbances of circulation, or of the collateral œdema, than the autopsy does. For instance, if paralysis and spasm accompany a disease of the cortical and medullary substance of the cerebrum, which does not encroach on the cranial cavity, and whose destruction does not induce paralysis and spasm, there is probably an anæmia extending to portions of the brain lying far deeper. As numerous examples have proved that an entire half of the cerebellum may be destroyed without inducing hemiplegia, we cannot refer a hemiplegia, observed along with structural changes confined to the cerebellum, directly to that organ, but must consider it due to the extension of collateral œdema to portions of the brain whose loss of function causes paralysis of half the body. The strange experience, that, in disease of one side of the cerebellum, there is sometimes no hemiplegia, at others there is hemiplegia of the same side, and in still other cases that it occurs on the opposite side, is doubtless due to the fact that, in the latter cases, a collateral œdema extends along the crura cerebelli ad frontem to the lateral regions of the pons; while, in those cases where the same side is affected, the œdema extends along the crura cerebelli ad medullam oblongatam to the lateral branches of the medulla oblongata; and where there is no hemiplegia the collateral œdema has not advanced in either direction to regions whose loss of function involves that symptom. These examples may suffice to show how important a rôle partial anæmia of the brain, due to collateral œdema, plays in the symp-

tomatology of brain-diseases confined to certain points. I have already remarked that the variation of symptoms, the temporary improvement or exacerbation, observed in the course of some brain-diseases, depend greatly on the increase or decrease of the collateral œdema about the point of disease.

The third form of partial anæmia of the brain, the result of compression of the capillaries of portions of the brain from diseases causing pressure, induces constant and characteristic symptoms, which of course vary according as the anæmia is in one of the greater hemispheres or is below the tentorium. If the capillaries of one of the greater hemispheres be compressed by an effusion of blood, by a tumor, or any other local disease contracting the space in the skull, there will be hemiplegia, no matter where the said disease be located. This hemiplegia is limited to the lower half of the face, and to the two extremities of the opposite side. It has often been considered as enigmatical, that in many cases diseases above or below one of the greater hemispheres, as well as within it, led to hemiplegia, while in other cases the same diseases at the base, convexity, or in the medulla of a great hemisphere did not induce hemiplegia; and tables have been made out which show at a glance this want of correspondence. I consider these tables as utterly worthless, unless the variety of the disease be stated in them; and I think it very important to distinguish between two classes of disease, whose effect is very different; namely, those which occupy more space than the brain-filaments and ganglion-cells which they supplant, and those which do not. Diseases at the base, convexity, or in the medullary portion of the cerebrum only induce hemiplegia when they lessen the space, in other cases they do not cause it (unless collateral œdema in their vicinity extend to the thalamus and corpus striatum). There are exceptional cases where hemiplegia does not occur in disease of one of the greater hemispheres whose products certainly contract the space. When tumors grow very slowly the brain usually atrophies, and as much space may be gained in the skull by the disappearance of brain-substance as is lost by the slow growth of the tumor. In such cases there is no anæmia of the affected hemisphere from compression of the capillaries, and consequently, if the tumor be not in the immediate vicinity of the corpus striatum and thalamus, hemiplegia does not occur. We must also remember the extensive communication between the two upper chambers of the skull at their anterior part. It is evident that at this place pressure on one hemisphere may more readily be propagated to the other than at any other part. But, the more pressure is divided up, the weaker its action becomes. In accordance with these considerations, diseases of the anterior lobes, which do not encroach too much

on the space, do not entirely compress the capillaries of the affected hemisphere, so that they do not cause hemiplegia, or at least it is only slight, while the propagation of pressure to the other hemisphere is shown by psychical disturbances, which are usually absent where the affection is limited to one side of the brain. Perhaps this may partly explain the aphasia, which is found with disease of the frontal lobe of one side, particularly (but not constantly) of the left side; since, in the region of the frontal lobes, pressure acting on one side is very readily propagated to the other. As the two sides of the brain are so symmetrical, it is difficult to believe that there is any organ in one which does not exist in the other.

Even more characteristic and more constant is the combination of symptoms accompanying compression of the capillaries of the parts of brain in the posterior cranial fossa. This is apparently because the tentorium can offer greater resistance to pressure acting on it than the falx can; also because the communication from the posterior and lower chamber of the skull, bounded by the tentorium and occipital bone, with the upper chambers, is far less free than that which exists between the two upper chambers. As is well known, we may readily err in diagnosis of diseases of the brain, but I do not remember to have made a mistake when I have given a diagnosis of disease contracting the space in the posterior cranial fossa. Many of my former pupils also have assured me that, from experience in their own practice, they must regard the diagnosis of diseases limiting the space in the posterior cranial fossa as easy, and that they have repeatedly diagnosed them according to my instructions, and have verified the diagnosis by autopsy. The combination of symptoms from which we may diagnose compression of the capillaries of the parts lying in the posterior cranial fossa is as follows: Pains in the back of the head, sympathetic vomiting, a peculiar dizziness, diminution of sensibility and motor power, but no complete paralysis and anæsthesia regularly spread over the body, and impaired articulation and deglutition. The pains at the back of the head doubtless proceed from the filaments of the trigeminus going to the tentorium. As the sympathetic vomiting occurs in various brain-diseases, it alone has no diagnostic value, but, in combination with other symptoms, it greatly aids to render the picture of the disease characteristic. The dizziness accompanying diseases contracting the space in the posterior cranial fossa is not a hallucination, a subjective sensation of movement of the patient's own body, or of surrounding objects, that does not really take place. Unlike this far more frequent hallucinatory form of dizziness, it does not occur while the patient is quietly lying or sitting down, but results from certain bodily movements. When a patient complains of dizziness,

we should make it a rule to ask at once whether the attacks come on during rest, or only during walking, rising, etc.; if the latter points be answered affirmatively, we have obtained an important diagnostic point. The dizziness observed in diseases encroaching on the posterior cranial fossa depend, as *Immermann* has fully proved, on actual movements of the body, which the patient does not perfectly perceive, but which only have a general influence on his feeling of equilibrium. In healthy persons, vibratory movements of the body would also occur in walking, rising, etc., if they were not prevented by contraction of the muscles which straighten and curve the spinal column. When a person is walking stiffly and uprightly, we may readily see, by the increased prominence of the bellies of these muscles, that he is unconsciously using them. This facility of limiting, by muscular action, the movements and vibrations conveyed to the trunk is very much impaired in persons with disease encroaching on the posterior cranial cavity, a circumstance which is apparently supported by the supposition that the cerebellum chiefly causes innervation of the body, and is the prop of the spinal column (*Griesinger*). The diminution of sensibility and that of motor power, which render the patient awkward and uncertain, without increasing to complete paralysis and anæsthesia, are simply explained by the fact that the nerve-filaments, passing from the cerebrum through the foramen occipitale superius, enter the posterior cranial fossa, again leave it by the interior occipital foramen, and are thus subjected to a compression which impairs the propagation of excitement from the brain to the motor nerves, and of peripheral excitement of the sensory nerves to the brain. In the disturbance of speech it may be readily seen that it does not depend on lack of thoughts, or on the impossibility of finding words for the thoughts, but on the uncertainty and clumsiness in executing the movements necessary for distinct, rapid, and connected articulation. The disturbance of deglutition is occasionally designated as difficulty of swallowing; it usually manifests itself by slight choking while drinking. I shall not attempt to decide whether this disturbance of articulation and deglutition depends on disturbance of the function of the hypoglossal and glossopharyngeal nerves at their origin, or in the nerves themselves, by diseases affecting the space in the posterior cranial fossa. Besides the above-mentioned symptoms, there are usually others depending on the injury of certain nerves, and, if the orifice of the venæ Galeni into the straight sinus be compressed, and the escape of blood from the ventricle prevented, we have the symptoms of chronic hydrocephalus. We shall hereafter speak of this when treating of tumors and abscesses of the cerebellum, pons, medulla oblongata, and of chronic hydrocephalus. Here will be the place to mention those symptoms which are

observed, with remarkable regularity, in diseases affecting the space in the posterior cranial cavity, no matter what their nature or location. Among my pupils I have noticed that the interest in brain-diseases rapidly increases when it becomes evident that, in many cases, a sure diagnosis can only be made within certain limits. Not unfrequently we are obliged to stop at the diagnosis of a disease encroaching on one side of the cerebrum, or on the posterior cranial fossa. In the following chapters we shall show under what circumstances we can go further, and how, in other cases, a certain diagnosis may be made of the nature and exact seat of the disease.

Treatment.—As may be readily understood, the treatment of partial anæmia and necrosis promises little benefit. In thrombosis and embolism of the cerebral arteries, the obstruction to the supply of blood cannot be removed by therapeutic remedies. Hence the indication would be to favor the development of a collateral circulation, without exposing the patient to new danger from too great collateral fluxion. It is very difficult to fulfil these indications, and we may readily do harm instead of good. The purer the symptoms of partial paralysis, the more obstinate they remain; if no symptoms of irritation accompany them, the more a strengthening and stimulant treatment is indicated. Hence we always find the administration of stimulants recommended in the treatment of softening of the brain. If, on the other hand, the symptoms of irritation caused by fluxionary hyperæmia, such as severe headache, contractions, etc., are prominent, it is advisable to use cold, and apply leeches behind the ears repeatedly. We should be careful about venesection, as it is readily followed by collapse. From what has been said, it is evident that we can give no general rules, but the treatment must be suited to each case. We shall speak of the treatment of partial anæmia of the brain, induced by collateral œdema and compression of the capillaries, when we treat of the diseases that this form of partial anæmia accompanies.

CHAPTER V.

CEREBRAL HÆMORRHAGE—APOPLECTIC STROKE—APOPLEXIA SANGUINEA.

By the term stroke, or apoplexy, was originally meant, as the word indicates, the sudden occurrence of complete functional inactivity of the brain. Apoplexy is divided into various forms, according as the paralysis is induced by an extravasation of blood or by a serous effusion, or as organic diseases can or cannot be found as the cause: thus we have Apoplexia sanguinea, A. serosa, A. nervosa, etc. In the present

chapter we treat of the lesion of the brain characterized by rupture of the blood-vessels and escape of their contents, whether it induces the symptoms of sudden paralysis of the brain or not.

Etiology.—Cerebral hæmorrhages almost always occur from the smaller arteries or the capillaries of the brain, and are caused partly by structural disease of the arterial walls, partly by an anomalous condition of the part of brain surrounding the vessels, partly by increased pressure of the blood against the wall of the vessel. The bleeding most frequently occurs when several of the factors act together.[1]

The structural changes in the walls of the vessels, to which their abnormal fragility is due in most cases, are the results of endarteritis deformans, which was treated of in the first volume. This explains the frequency of apoplexy in persons over forty years of age, which was noticed even by *Hippocrates*. Next to this, simple fatty degeneration of the arterial walls, not dependent on inflammation, but occurring in badly-nourished cachectic and chlorotic persons, also induces greater fragility and ruptures of the cerebral vessels. Still we must say that fatty degeneration of the finer cerebral arteries is found far more frequently than would be expected from the proportionately rare occurrence of apoplexy. Occasionally rupture of the entire arterial wall is preceded by rupture of the inner and middle coats, while the adventitia still resist. In such cases the blood escapes between the external and middle coats, and small dissecting aneurisms are formed. Lastly, there are cases where abnormal weakness of the cerebral vessels must be supposed, although it cannot be proved. These are the rare cases where cerebral hæmorrhages are found in convalescents from typhus and other acute infectious diseases and during scorbutus.

We have already mentioned that, in necrotic softening of the brain, capillary hæmorrhages not unfrequently occur along the borders of the softened part. Frequently, gradual atrophy of the brain causes dilatation and final rupture of the vessels. While the brain-substance disappears, a vacuum cannot form in the skull; hence increase of the cerebro-spinal fluid and dilatation of the vessels are necessary results of senile or any other form of atrophy of the brain, which is a frequent sequel of the most varied forms of disturbance of nutrition. Perhaps the frequency of apoplexy in advanced age depends at least partly on this circumstance, and there is no doubt that the atrophy of the brain, which is in many cases caused by the first apoplectic attack, has something to do with the frequent recurrence of apoplexy.

The increased pressure of the blood on the walls of the vessels, by which the latter are ruptured, may depend on any of the causes which we indicated in the first and second chapters as causes of hyperæmia. The frequent occurrence of apoplexy during long and luxurious meals

tends to show that the hyperæmia of the brain induced by temporary plethora is one of the most dangerous forms. Hypertrophy of the left ventricle, particularly that form resulting from any extensive endarteritis deformans, plays an important part in the ruptures of cerebral vessels. In the latter case two dangerous factors unite—the morbid fragility of the vessels and the increased pressure of the blood on them. Moreover, small arteries, in which there is otherwise a regular pressure of the blood, and whose walls also maintain a nearly equal tension during the systole and diastole of the heart, pulsate when there is extensive atheromatous degeneration, and at every systole of the heart the normal medium tension of their walls is decidedly increased. It will be readily understood that this circumstance also increases the liability of the vessels to rupture. Cerebral hæmorrhages so often depend on the complication in question, that, in doubtful cases, the discovery of hypertrophy of the left ventricle and of an atheromatous degeneration of the arteries may decide the diagnosis.

Apoplexies occur at all times of the year; occasionally, without any known cause, cases accumulate remarkably. They have also been observed at all times of the day, and statistical tables have been made of their comparative frequency at morning, mid-day, and evening. Although advanced age furnishes the largest number of cases, apoplexy does occur even among children. Men are somewhat oftener attacked than women. There is no such thing as an apoplectic constitution, indicated by a short neck and broad shoulders.

Anatomical Appearances.—The distinction is made into capillary hæmorrhage and hæmorrhagic clots, according as the bleeding consists of numerous small, closely-packed effusions, or of a larger quantity of blood.

In capillary hæmorrhages the cerebral substance appears dotted with dark-red punctate extravasations at some point of variable size. The cerebral substance between the small extravasations either retains its normal color and consistence, or is colored yellow or reddish to a variable extent by imbibition; it is relaxed and moist, or, lastly, it is broken down to a red pulp by the extravasation (red softening).

Small hæmorrhagic spots sometimes press the brain-filaments apart; but larger ones break up and become mixed with the brain-substance. In the former case the effusion is sometimes elongated in the direction of the filaments; in the latter it is more roundish or irregular. In the former case the walls of the effusion are to some extent smooth; in the latter they oftener appear ragged, and are frequently surrounded, for a space some lines thick, by a broken-down pulpy brain-substance discolored with blood. The size of the clot varies from that of a hemp-seed to that of the fist. If it be in the vicinity

of a ventricle, it often breaks through the wall of the latter, and its blood escapes into the ventricle. Hæmorrhagic effusions, lying near the surface of the brain, not unfrequently break through the cortical substance and pia mater, so that the blood enters the subarachnoid space. Usually there is only one hæmorrhagic effusion in the brain, rarely several. Their most frequent locality is the corpus striatum, the thalamus opticus, and the large medullary masses of the hemispheres; less frequently they occur in the cortical substance of the cerebrum, in the cerebellum and pons. Apoplexies in the corpora quadrigemina and medulla oblongata are rare, and they hardly ever occur in the corpus callosum and fornix. The contents of a recent apoplectic clot consist of blood and broken-down brain-substance. The blood either remains fluid or is partly coagulated, and then the fibrin is occasionally deposited at the periphery, while the middle of the clot consists of fluid blood. Changes of the contents and walls of the clot soon begin. The fibrin of the blood and the portions of brain mingled with the effusion break down into a detritus, the contents become more fluid, the dark-red color becomes brown, then saffron yellow. Granular pigment and often also hæmatoidin crystals are formed from the hæmatin. At the same time, in the immediate vicinity of the clot, there is a new formation of connective tissue starting from the neuroglia, which develops into a thick, hard layer that encloses the clot. In the same way there is a new formation of delicate connective tissue, colored yellow by the pigment contents and serous infiltration, which covers the walls and traverses the clot as a fine network. After the elements of the effusion have broken down, all its remains disappear, while their place is gradually supplied by serum; and then we find in the brain a cavity filled with clear liquid, surrounded by a callous substance, and covered and traversed by delicate yellow-colored connective tissue—an *apoplectic cyst*. The cysts usually remain permanently. But occasionally the serum is reabsorbed, the walls approximate, and finally are only separated by a stratum of pigment. These callous spots, enclosing pigment striæ, are called apoplectic cicatrices. The cicatrization of a hæmorrhagic effusion in the cortical substance is somewhat different. The effusion of blood under the pia mater, which is usually flat and extended, undergoes the same changes as do the contents of central clots. The red pulp gradually becomes a reddish-brown or saffron-yellow crumbly mass, which is bounded below by callous brain-substance, above by the pia mater. Lastly, we find an excavated pigmented plate, above which a serous effusion fills the cavity resulting from the depression. While the above terminations of cerebral hæmorrhages must be regarded as the most favorable, in some cases the reactive inflammation in the vicinity of the broken

down portion of brain is not limited to a new formation of connective tissue, but there is extensive destruction from inflammatory softening, or the apoplectic clot is even transformed to an abscess of the brain. The parts untouched by the hæmorrhage are bloodless if the extravasation has been considerable, and the more so the larger the extravasation; small capillary hæmorrhages have no influence on the amount of blood in the rest of the brain. In large effusions the hemisphere containing the clot is particularly anæmic. The subarachnoid spaces also are empty, the convolutions flattened, and the furrows have disappeared. Since the uneven appearance of the surface of the brain is to a great extent due to the presence of cerebro-spinal fluid and of vessels containing blood between the convolutions, the surface of the brain appears very smooth and even when there is a large extravasation. The brain rarely endures the injury from an apoplexy without impairment of the general nutrition. The gradual diminution of mental power seen in almost all apoplectic cases corresponds to a general atrophy of the brain; which, according to the examinations of *Türk*, is associated with a degeneration, extending into the spinal medulla, of those filaments communicating with hæmorrhagic effusion.

Symptoms and Course.—Sometimes apoplexy occurs unexpectedly in persons who have previously felt quite well; in other cases it is preceded by premonitory symptoms that excite in the physician, and even in the laity, the apprehension that the patient is threatened with apoplexy. The patients complain of headache, or of heaviness and fulness of the head, of noise in the ears, flashes before the eyes, and attacks of dizziness; they sleep badly, are excited and irritable. There are also, as peculiarly ominous symptoms, occasional temporary feelings of formication and numbness in certain limbs, momentary loss of memory for some words and figures, or temporary paralysis confined to certain groups of muscles. Thus we see the premonitory symptoms of apoplexy are those that we gave in preceding chapters, as due to general and partial hyperæmia of the brain, and to partial anæmia of the brain resulting from degeneration of the walls of the vessels; a condition that entirely corresponds with the frequent dependence of cerebral hæmorrhages on increased lateral pressure in the cerebral vessels, and on degeneration of their walls. In general it cannot be determined whether the temporary partial paralysis and anæsthesiæ, the "warnings" preceding the apoplectic attack, depend on thrombosis of small vessels, or on small capillary hæmorrhages. In the latter case, of course they could not be classed among premonitory symptoms.

The destruction of brain-filaments, whether they be broken up by large extravasations or be softened by small capillary hæmorrhages,

can, as we have often shown, have no other direct result than partial paralysis. Since the destruction of certain portions of the brain, particularly in the medulla of the cerebrum, does not cause any perceptible disturbance of function, we may readily understand that small hæmorrhages, as well as other diseases of those parts, may occasionally escape recognition during life.

We have designated the corpus striatum and thalamus opticus as the most frequent seat of hæmorrhage; a destruction of these parts, or of the pedunculi cerebri, induces paralysis of the opposite half of the body. We may readily determine that the paralysis, resulting from destruction of the above parts, only depends on interruption of the conduction between the organs acting in thought and will and the motor nerves and muscles; the power to think and will is unimpaired. After the apoplectic seizure, during which there is complete loss of consciousness, has passed off, if we ask the patients to give us the affected hand, they show their desire to fulfil the request, by taking the paralyzed hand in the other one, whose nerves and muscles are under the control of their will, in order to accomplish the act. On the other hand, in recent cases, every motor nerve on the paralyzed side, to which we apply the induced current, causes contraction in the muscles supplied by it. Hence the only failure is in the communication between the central excitory apparatus and the motor nerves. This interruption has no effect on those movements of the paralyzed side that occur in a reflex manner without the influence of the will; for patients, who, as a result of apoplexy on the left side, cannot move the right arm or leg, move the right side of the thorax just as well as the left during respiration. And the connection between the motor nerves and those nerve-filaments and ganglion-cells which are excited during certain states and feelings of the mind is not always removed with the interruption of conduction at present under consideration. This is shown by the fact that some patients who cannot make the motions of laughing or crying on the one side of the face, at our request, can do so when they do not *will* to do it, but when their feelings lead them to do so. In the same way, the interruption of conduction of the impulses of the will, from the central organs to the motor filaments, does not necessarily imply that the communication of the latter with sensory and with other motor filaments is interrupted. On the contrary, we find that occasional reflex movements sometimes remain undisturbed in the paralyzed parts, or even occur more readily, so that it appears as if, when the excitement of the motor filaments is no longer subject to the will, it occurs more readily than it otherwise would. Paralysis of half the body, due to destruction of the corpus striatum and thalamus of one hemisphere, is characterized by its limitation to the muscles of

the extremities, to those muscles of the face going to the angle of the mouth and the nose, and to the muscles that protrude the tongue. The patients can almost always chew normally on the affected side, they can wrinkle the forehead, open and close the eyelids, move the eyes in any direction, etc. On the other hand, the patient frequently cannot lift the paralyzed arm and foot an inch from the bed; the mouth hangs down on the affected side, and the nostril is contracted, occasionally the cheek flaps about like a loose sail at every expiration, while, on the sound side, the angle of the mouth is drawn up and the nostril dilated. If the patient protrude the tongue, its point goes toward the paralyzed side, because only the muscles of the opposite side push forward the root of the tongue and elongate that organ. In most cases, along with the hemiplegia, there is also anæsthesia of half the body, but after a time this usually passes off partially or entirely. This course of the anæsthesia, as well as the experience that animals have no sensation after destruction of their corpus striatum and thalamus, and that, after taking them away, the power of feeling peripheral pain continues, appears to indicate that the temporary anæsthesia of the paralyzed half of the body does not depend immediately on destruction of the corpus striatum and thalamus, but on the compression of the capillaries in the sections of brain lying below them, caused by the effusion of blood.

The same symptoms as are caused by effusions of blood into the thalamus and corpus striatum, are induced by effusions at other parts of the cerebrum, provided they are extensive enough to compress the capillaries of the thalamus and corpus striatum. After the discussion in the previous chapter concerning partial anæmia of the brain and its influence on the cerebral functions, this similarity cannot appear strange to us, but must rather be regarded as evident and necessary. The only difference is the following: A large apoplectic clot, destroying the corpus striatum or thalamus, leaves a hemiplegia that never disappears; only small clots in these parts, by which the filaments and ganglion-cells are not broken down, but only pressed apart, leave paralysis which is occasionally temporary. Hence we may conclude that the apparatus for exciting the motor nerves, which doubtless exists in the brain, although it may itself be excited by the will, is located in the vicinity of the corpus striatum and thalamus. On the other hand, extensive apoplectic clots at other parts of the cerebrum not unfrequently leave paralysis, which sooner or later disappears again. From this course we may suppose that the capillaries of the motor centres, being relieved of the pressure by the partial resorption of the extravasation, have again become permeable to the blood; or that the collateral œdema in the vicinity of the broken-down part of brain, which

extended to the motor centres, has disappeared with the cicatrization of the apoplectic clot.

Hemiplegia occurs in some, but not in all, of the cases where the effusion is into the cortical substance of the cerebrum; these are generally accompanied by hæmorrhage into the tissue of the pia mater. The difference, doubtless, depends on how far the frequently-mentioned results of the hæmorrhage, compression of the capillaries, or collateral œdema, extend inward; whether they reach the corpus striatum and thalamus or not. When the clot is in this position, general convulsions are often observed, and in most cases there is severe disturbance of the psychical functions. Since experience shows that persons with very advanced and extensive degeneration or atrophy of the cortical substance (if it be only on one side) often have no psychical disturbance, the frequent occurrence of the latter in apoplexy on one side is probably because hæmorrhages readily affect the other hemisphere; and particularly because this is likely to occur in the complicating inflammation of the pia mater, which has a great tendency to spread.

Hæmorrhages in the pons, if of any considerable size, and in the medulla oblongata, even if very slight, usually cause death. In small effusions in the lateral portions of the pons, there are anæsthesia and paralysis on the opposite side of the body; in small extravasations in the middle of the pons, there is paralysis of both sides.

In hæmorrhages of the cerebellum we often find paralysis of the opposite side. But this cannot depend on the affection of the cerebellum, for there is often no paralysis observed when this is extensively destroyed.

We must not think that, because hæmorrhages are found at very different parts of the brain, the different cases of paralysis dependent on cerebral hæmorrhages will differ widely from each other. On the contrary, the large majority of cases show a great similarity, as they induce the hemiplegia above described. It is, of course, of great practical importance to know this, which is simply explained by the fact that, according to statistics, seven-eighths of all cerebral hæmorrhages are located in the cerebrum and particularly in the vicinity of the corpus striatum and thalamus. There are some very wonderful exceptions to the one-sidedness of the paralysis and to its occurrence on the opposite side, in hæmorrhage of the cerebrum, which we are at present unable to explain satisfactorily. However, we must add that, of late, since all anomalies existing with the hæmorrhage, particularly plugging of the cerebral arteries, are more carefully attended to, and used in explaining the symptoms, the number of such cases published has greatly decreased.

Another series of symptoms in cerebral hæmorrhage, which is

called a *stroke* of apoplexy, and is only absent when the hæmorrhage is slight, do not depend directly on the local injury of the brain, but on its effect on the rest of the brain. We shall hereafter show that the apoplectic fit usually occurs at the outset, but sometimes does not come on till after the appearance of the paralysis. The stroke of apoplexy rarely develops gradually and probably only does so when the hæmorrhage occurs slowly; in most cases it comes on suddenly, and the patient falls to the earth (often with a cry) as if "knocked down." During the attack, consciousness is entirely lost, as well as the power of feeling and moving. The sphincters also are generally paralyzed, so that fæces and urine are passed involuntarily. Respiratory movements alone, which depend on the medulla oblongata, are continued; but the inspirations follow at long intervals, and are usually loud and stertorous, since the paralyzed and pendulous soft palate is thrown into vibration by the air. The patient has a peculiar appearance, from the relaxed cheeks being puffed out with every expiration. There is often vomiting at the commencement of the attack; the pulse is very slow, the pupils contracted.

It is usually supposed that the apoplectic fit is a result of the pressure or bruising of the nerve-filaments and ganglion-cells of the entire brain by the extravasation. However, it is evident that this pressure can never exceed that of the blood in the cerebral arteries; for, as soon as the pressure in the parts around the arteries is as great as that of the blood in the vessels, no more blood can escape from the latter. But, from experiments that we can make on peripheral nerves, there is no doubt that such a pressure is entirely insufficient to annul the excitability of the nerve-filaments. The following fact also tends to disprove the current explanation: If the symptoms of paralysis depended on the pressure to which the filaments of the brain are subjected in apoplexy, bleeding should remove these symptoms not only in some but in all cases, provided enough blood be drawn to lessen the pressure in the whole vascular system, particularly in the arteries. *Hyrtl*, who also speaks most decidedly against referring these symptoms to pressure on the brain, thinks that the accidents generally referred to "pressure on the brain" are due to a slight amount of concussion; but, apart from the fact that there is no such concussion in cases of extravasation which are not of traumatic origin, no anatomical changes referable to such a cause can be found. We refer the apoplectic fit to sudden compression of the capillaries, that is, anæmia of the brain-substance. In all large hæmorrhages this anæmia may not only be recognized with certainty after death, but even during life it shows itself by a very important symptom which is usually falsely interpreted, that is, by a remarkable pulsation of the carotids. This

symptom is very generally regarded as a sign of "increased pressure of blood to the head," although it really indicates that the flow of blood into the skull is obstructed; we may at any moment induce the same phenomenon in the artery of the finger by tying a string tightly around the end of the finger. All diseases of the brain and its membranes affecting the space in the skull enough to obstruct the afflux of blood from the afferent vessels, hence not only large effusions of blood, but also abundant exudations and transudations, large tumors, etc., are accompanied by increased pulsation of the carotids. If we find this symptom, when there is no hypertrophy of the left ventricle, nor corresponding pulsation in other arteries, it will, in doubtful cases, be a great aid to the diagnosis of some brain-disease encroaching on the cranial cavity. In my clinic my pupils have frequently had an opportunity of satisfying themselves of the correctness of the indication and of the great diagnostic value of this symptom. If we regard the physical conditions, we see that anæmia, at all events arterial anæmia, of the brain can never result from rupture of the capillaries; for the escape of blood from the ruptured capillaries can only last till the tension of the contents of the skull equals that of the blood in the capillaries. In accordance with this we see that the apoplectic fit does not occur in capillary hæmorrhage. If, on the other hand, an artery be ruptured, and the bleeding does not soon cease from some other cause, the tension in the surrounding parts finally becomes as great as it is in the artery; and, as it is greater there than in the capillaries, the latter must be compressed and become impassable for the arterial blood. In accordance with this, apoplectic fits almost always accompany arterial hæmorrhages of any extent. If we analyze the above symptoms of the apoplectic fit, we find that, while it lasts, the functions of both sides of the cerebrum are lost. The patients have no feeling, even on the strongest peripheral irritation; they cannot make the slightest motion, and consciousness is entirely lost. On the other hand, those portions of the brain through which acts indispensable to life, especially respiration, are performed, retain their functional power. This is, apparently, because the falx protects the opposite hemisphere less, from the compression of the capillaries by the extravasation, than the tentorium does the medulla oblongata. Effusions of blood below the tentorium, even when slight, are very dangerous, because in them the medulla oblongata is not thus protected, and its functions are readily disturbed by compression of its capillaries. I shall not attempt to decide whether or not the slowness of the pulse, the diminished frequency of respiration, and the contraction of the pupil, observed during apoplectic fits from effusions above the tentorium, are due to increased excitement of the vagus and oculo-

motor nerves as a result of pressure acting on them, but somewhat modified by the tentorium.

If the patient does not die during the apoplectic fit, but recovers consciousness, he shows signs of a more or less severe encephalitis in a few days. This depends on the injury to the brain from the hæmorrhage, hence must be regarded as traumatic. When it does not reach a great height, and only leads to new formations of connective tissue about the clot, the symptoms are increased frequence of pulse and other signs of fever, headache, sparks before the eyes, delirium, occasionally also twitchings and contractions of the paralyzed parts. After a time these "symptoms of reaction" moderate and finally disappear, and the patient is well except for the remaining paralysis. But, if the inflammation in the vicinity of the clot is of considerable intensity and induces inflammatory softening, the above symptoms are accompanied by those of general paralysis, and the patient dies as a result of too great severity of the so-called symptoms of reaction.

[Hemiplegia caused by cerebral apoplexy shows the peculiarities of all cerebral palsies (as contrasted with spinal and peripheral palsies). The loss of sensation is less marked and more transitory than that of motion, and the reflex action is almost always preserved or even increased through irritation from the apoplectic centre. Hence a slight increase of electrical irritability argues in favor of a cerebral palsy. The arm is usually more affected than the leg, and, for a time at least, the tongue and facial muscles are often likewise involved. Cerebral palsy of the face especially affects the nervous twigs which supply the muscles of the alæ nasi, those of the angle of the mouth, and the buccinator muscles; while the nerves of the orbicularis palpebrarum almost always escape, so that the eye can always be closed (in contrast to what occurs in peripheral facial palsy). A facial palsy of cerebral origin is upon the same side as that of the hemiplegic limbs; whereas peripheral palsy of intracranial origin (like that by compression of the facial nerve along its course in the base of the skull) is upon the side where the lesion exists. There are often vasomotor disturbances, and the paralyzed side (*Eulenburg*) shows by the sphygmograph that the arterial tone is sunken and the current retarded, and that the temperature is lower than upon the healthy side; moreover, the hand and foot are often swollen. The psychical functions also are apt to be more or less altered.

The derangements which remain after an apoplectic attack has abated may continue to improve for weeks and months; but where a perfect recovery does not promptly take place, it seldom does so

afterward, so that a paresis of the arm and leg is apt to remain. The palsied limbs often move sympathetically, particularly during the acts of coughing and sneezing. Several weeks after the fit there generally occurs a gradually increasing contraction of the crippled limbs (of the hand and arm especially). Thus all the joints of these members become permanently flexed, and this may go so far as to require that the palm of the hand be protected from the impression of the finger-nails. This change from flaccidity to stiffness is an evil prognostic sign, meaning that the case is incurable.

Arthropathy of the hemiplegic, described by *Scott Alison*, *Brown-Séquard*, and *Charcot*, is confined to the palsied limbs, and prefers the arm, and especially the shoulder. This complication, like the contractions, appears several weeks after the fit, and the pain, redness, and swelling are sometimes so marked that the attack might be mistaken for acute articular rheumatism, did not the seat of the disease upon a paralyzed limb indicate its true significance.

A very rare sequel of apoplexy is a permanent hemi-anæsthesia, which persists even after the motor derangement has been restored. This anæsthesia may be complete or incomplete; it involves not merely the skin, but the deeper parts of the lip, tongue, and palate.

The consequences of an apoplectic stroke, however, are by no means always alike. The situation of the clot may affect the character of the symptoms. While a small extravasation upon the cortical substance may cause no recognizable symptoms, a large one, or numerous extravasations in the cerebrum, would be apt to cause mental derangement. There may be stupor, convulsions, delirium, mania, and permanent insanity. Large clots in the posterior brain may cause paralysis or prove speedily fatal.

Hæmorrhagic, embolic, and other lesions affecting the third frontal convolution, particularly those of the left anterior lobe, cause the well-known disturbance of speech known as aphasia, of which more hereafter. Numerous dissections would seem to show that the whole region involving the fossa Sylvii, the island, and the corpora striata may be similarly affected by injuries. Apoplexy of the crura cerebri is said to be characterized by marked anæsthesia, slight facial palsy, and squinting, while the psychical functions remain unchanged.

Apoplexy may result in paraplegia when the lesion is bilateral. In such cases the two sides are usually affected in unequal degree. This very rare consequence of cerebral apoplexy is to be distin-

guished from paraplegia of spinal origin by the presence of mental derangement or of palsy of one of the facial cranial nerves; it being borne in mind, however, that cerebral and spinal palsies may coexist.

When an apoplectic effusion bursts its way to the meninges or into one of the ventricles, death usually ensues. As a rule, such penetrations are attended by convulsions, and by the very early appearance of contractions upon the palsied side, sometimes even upon the other. There are also epileptiform fits. Apoplexies of small extent often are purely local in their effect, causing paresis of a single extremity, thickness of speech, facial palsy without hemiplegia, sudden amaurosis or amblyopia of one eye, ptosis, etc. Such small extravasations may form without the symptoms of a fit, so that doubts as to the real nature of the disease may exist until a subsequent grave apoplectic stroke clears up the mystery.]

From the varied size and number of the hæmorrhages, their different seats, the greater or less rapidity of their occurrence, as well as from the more or less severe inflammation of the surrounding brain, we have many varieties in the course of cerebral hæmorrhages, of which we shall mention only the more important.

A rather frequent appearance of the disease, which corresponds to a rapidly-occurring extensive hæmorrhage, or to the coincident occurrence of several hæmorrhages, is the following: After some premonitory symptoms, or even without them, an apoplectic fit suddenly occurs; the patient does not again recover consciousness, the paralysis extends to the medulla oblongata, the breathing becomes irregular, the pulse intermittent and slow, the pupils dilated, and death occurs in a few minutes (apoplexie foudroyante), or after some hours.

In a second class, which is most frequently seen, and which is also due to a rapidly-occurring but probably less extensive hæmorrhage at the usual places, there is also first an apoplectic fit; even during unconsciousness we may see by the distortion of the face, by the peculiar relaxation of the muscles on one side, sometimes also by the dilatation of one pupil, which is the paralyzed side. After a few minutes or some hours, or occasionally not till next day, the patient gradually arouses from the stupor; but he speaks indistinctly, and hemiplegia, with the peculiarities above described, becomes evident. On the second or third day there are fever and the other symptoms of traumatic encephalitis. After these have disappeared, the patient remains for the rest of his life paralyzed on one side of his body, although the part of the paralysis due to œdema in the vicinity of the apoplexy disappears after a time.

In other cases, most probably when the hæmorrhage ceases for a time and returns again, and continues moderately, the disease begins with an apoplectic fit, from which the patient arouses after a time. We note the hemiplegia, but hope that this time the patient will escape with his life. But, after a few hours, consciousness again begins to disappear, and finally is lost entirely, does not return, and the patient dies comatose.

A slowly-occurring hæmorrhage, which finally becomes very abundant, appears to induce the cases where an apoplectic fit does not open the scene, but where there is first hemiplegia, and, later, loss of consciousness and general paralysis of the brain.

It would require too much time to fully describe other appearances, particularly the modifications resulting from the different severity of the reactive inflammation in the vicinity of the clot, and from the varied grades of the consecutive atrophy of the brain.

Treatment.—For the prophylaxis of apoplexy, we may refer to Chapters I. and IV. of this section, since the prophylaxis of cerebral hæmorrhage demands the same general rules that we gave for the treatment of cerebral hyperæmia, and for the disturbances of circulation resulting from disease of the arteries. If a patient has had one attack of apoplexy, he must be particularly careful to avoid all causes by which the cerebral vessels may be overfilled and distended; he must especially avoid long, luxurious meals, and must keep his bowels regular.

If cerebral hæmorrhage has occurred, it becomes our object to prevent a continuance of the bleeding, to induce reabsorption of the extravasation, and the formation of an apoplectic cicatrix. But we must not deceive ourselves as to our power, and must understand that we have no remedy for arresting the hæmorrhage, or for hastening the reabsorption and cicatrization. In the treatment of this disease we are restricted to combating the more dangerous symptoms as well as possible. Not a few patients, in apoplectic fits, recover consciousness during venesection, and it seems as if we could, not unfrequently, prevent the extension of the paralysis from the cerebrum to the medulla oblongata, which is indispensable to life, and so save the patient by bleeding. On the other hand, there is no doubt that, in many cases, bleeding during an apoplectic fit hastens a fatal result; collapse occurs immediately after the venesection, and the patient never arouses. We have previously said that bleeding must always prove beneficial, if the symptoms given as signs of pressure on the brain were actually induced by the pressure to which the brain is subjected by the extravasation; and we have also said that the want of success in venesection, in many cases, spoke against this explanation. From the explanation

that we have given of the apoplectic fit, it is evident that, under some circumstances, venesection is a very useful remedy; under others it is very injurious, and the indications for it may be very exactly given. In order that as much arterial blood as possible may enter the brain, we must try to facilitate the escape of the venous blood, without, however, diminishing the propelling power too much. If the impulse of the heart be strong and its sounds loud, if the pulse be regular, and no signs of commencing œdema of the lungs exist, we should bleed without delay. Local bleeding by leeches, behind the ears, or to the temples, or by cups to the back of the neck, cannot replace general bleeding, but may be used as adjuvants. If, on the contrary, the heart's impulse is weak, the pulse irregular, and rattling in the trachea has already begun, we may be almost certain that bleeding would only do harm, since the action of the heart, which is already weakened, would be still more impaired, and the amount of arterial blood going to the brain would thus be still more decreased. When the latter state occurs, the symptomatic indications require just the contrary treatment, in spite of the original disease being the same, and being due to the same causes. We must strive with all our skill, by the use of stimulants, to prevent paralysis of the heart. If we cannot give wine, ether, musk, etc., internally, we should apply large sinapisms to the chest and calves of the legs, rub the skin vigorously, sprinkle the breast with cold water, or drop melted sealing-wax on it.

If the patient has recovered consciousness after the apoplectic fit, we simply prescribe a mild, unirritating diet, keep the bowels open, and cover the shaved head with cold compresses, so as to prevent, if possible, too severe inflammatory reaction. According to the severity of the inflammatory symptoms which, nevertheless, occur, we may continue this simple treatment, and at most give a purge, or apply leeches behind the ears, and repeat the application if necessary. In this stage venesection is superfluous and dangerous. On the other hand, especially when the fever-symptoms have moderated, good is done by derivatives to the nape of the neck, such as blisters and pustulating ointments, which subsequently are no more to be used.

If the stage of inflammatory reaction has happily passed, and the patient is pretty well, except the paralysis, we should avoid prescribing strychnia and other remedies, which are neither theoretically nor practically useful, but should regulate the diet and bowels, and place the patient under the best possible hygienic influences. Well-to-do patients may be sent to Wildbad, Gastein, Pfäfers, or Ragatz. We must not hope that the destroyed filaments of the brain will be restored by the use of these waters, but experience shows that, at these places, both cerebral and spinal paralysis often improve; probably this im-

provement is due to the favorable influence of the baths on the inflammation about the clot, and on that portion of the paralysis due to it.

Lastly, it cannot be denied that paralyses are generally improved by the employment of the induced current of electricity. This is doubtless solely because "faradisation localisée" is one of the most powerful means of therapeutic gymnastics. After paralysis has lasted some time, its degree almost always depends partly on diminished excitability of the nerves, and on commencing atrophy of the muscles from long disuse. For both of these states the methodical excitement of the nerves by the induced current is certainly the best remedy, and, at all events, it deserves the preference to irritating liniments, salves, and tinctures.

CHAPTER VI.

HÆMORRHAGES OF THE CEREBRAL MEMBRANES—APOPLEXIA MENINGEA—HÆMATOMA OF THE DURA MATER.

Etiology.—Excepting traumatic hæmorrhages of the meninges, among which are to be classed those occurring during birth, this is a rare affection. Effusions of blood in the subarachnoid space, or between the dura mater and arachnoid, result mostly from the breaking through of a cerebral hæmorrhage. Occasionally, ruptures of aneurisms, or of degenerated arteries, cause the meningeal bleeding; in other cases, the cause cannot be found.

The extensive capsulated collections of blood occasionally found, on autopsy, on the under surface of the dura mater, are not, according to *Virchow's* instructive examinations, as was formerly supposed, simple extravasations of blood, at whose periphery the fibrin has been precipitated and the fluid part capsulated, but they are the remains of chronic inflammations of the dura mater (pachymeningitis), with hæmorrhagic exudations. *Virchow* calls this blood-sac hæmatoma of the dura mater. The blood filling it comes from the numerous large and thick-walled capillaries that have formed in the pseudo-membrane of the dura mater during this variety of chronic inflammation, and it has been effused between the layers of the pseudo-membrane. The causes of chronic pachymeningitis, with hæmorrhagic exudation, are not perfectly known. The disease occurs chiefly in old age, and remarkably often in persons with mental disease, and in drunkards. It appears to develop sometimes as an independent, sometimes as a secondary disease, due to injuries of the brow. In the latter case it is said that years may intervene between the injury and the first symptoms of hæmatoma (*Griesinger*).

Anatomical Appearances.—If the blood be effused in the sub

arachnoid space, we usually find it as a more or less thick layer spread over the surface of the cerebrum and cerebellum. If the arachnoid be not torn, of course we cannot wash off the extravasation by turning a stream of water on it. Usually part of the extravasation reaches the ventricles, and there also we find more or less blood. In hæmorrhages between the dura mater and arachnoid the extravasation is generally collected, more particularly on the tentorium and at the base of the skull, and thence extends into the vertebral canal. But on the convexity of the hemispheres we also find bloody masses, which may be washed off by squeezing out the sponge over them. In both forms of hæmorrhage there is either simply flattening of the convolutions and anæmia of the brain-substance, or else the layers of brain-substance next to the hæmorrhage are suffused with blood and softened.

Hæmatoma of the dura mater is usually located near the sagittal suture and has the form of an oval flat sac, which may attain considerable size, and may be four or five inches long, two or three broad, and half an inch thick. The walls of the sac are colored rusty-brown by altered hæmatin; its contents are partly fresh fluid or coagulated blood, partly dirty reddish-brown clots, that are unmistakably older. The corresponding half of the cerebrum is flattened, or even shows a depression. Not unfrequently the hæmatoma is on both sides. We have the opportunity, rather frequently, of observing the commencement of a pachymeningitis hæmorrhagica; for, in many autopsies, we find a delicate yellow or brown connective tissue, larger on the inner surface of the dura mater and firmly adherent to its surface.

Symptoms and Course.—Hæmorrhages in the subarachnoid space or on the free surface of the arachnoid, do not belong to the "local" but to the "diffuse" diseases of the brain. Hence, when there is no complication with cerebral hæmorrhage, they are not accompanied by the regional symptoms characteristic of this, especially hemiplegia; on the other hand, the apoplectic fit is usually uncommonly severe, as the bleeding is generally very abundant, and is spread over both sides. Frequently the apoplectic fit occurs suddenly without any premonitory symptoms, and the patients die with the above-described symptoms of apoplexie foudroyante. When this occurs, we can, at most, make only a probable diagnosis, which depends solely on the absence of hemiplegia, the symptoms of which may, as a rule, be distinguished, even in the severest fits, from cerebral hæmorrhage. In other cases the apoplectic stroke is preceded by severe headache, and in some cases by general convulsions. Since these symptoms, particularly the latter, only occur exceptionally in cerebral hæmorrhage, and are often seen in extensive disease at the convexity of the hemisphere, they, in connection with the absence of all signs of hemiplegia, enable us to decide

with greater certainty that the case is not one of cerebral but of meningeal hæmorrhage.

Hæmatoma of the dura mater often runs its course with symptoms from which the disease cannot be certainly diagnosed, and when it occurs in the course of mental affections, as so often happens, we cannot usually make even a probable diagnosis. In other cases, the following factors, to which *Griesinger* has called attention, enable us, with more or less assurance, to make a diagnosis of hæmatoma of the dura mater; if circumscribed headaches, gradually increasing to great severity, in the vicinity of the vertex and forehead be the first, and, for a long time, the only trouble of which the patients complain; and if, between the appearance of these pains and that of other severe brain-symptoms, there be an interval not so short as in acute diseases of the brain and its membranes, but shorter than in most chronic diseases of these parts, particularly in the different cerebral tumors, the first suspicion falls on inflammation of the meninges, particularly of the dura mater, since inflammation of the other membranes has so great a tendency to spread, that it is accompanied by diffuse, not by circumscribed, headache. We are the more justified in this, as the form of pachymeningitis in question occurs just at the point where the patients complain of pain. If the patient had been mentally diseased before the commencement of the headache, or given to drinking excessively, or if he had had an injury of the head, particularly of the forehead, some time previously, there is still more reason for supposing the case one of pachymeningitis, as is evident from the etiology. But we also know that this form of meningitis usually leads to a large effusion of blood, encroaching on the cerebral cavity, and that then the effusion is capsulated on one or both sides of the sagittal suture. Hence, if the headaches be subsequently accompanied by the signs of compression of the capillaries of the cerebrum, by mental disturbances, loss of memory, diminished power of thought, increased inclination to sleep, which finally increases to coma, a slowly-developing and usually not pure hemiplegia, after excluding various brain-diseases, we must think of hæmatoma of the dura mater as being in the first rank of those that may possibly be present. Since, in hæmatoma of the dura mater, there may be reabsorption of the blood and consequent freedom of the brain from the pressure on it, a favorable course of the disease and recovery of the patient speak for hæmatoma in doubtful cases. If the effusion of blood does not take place gradually, as in the course of the disease above described, but occurs suddenly; if it is large and limited to one side, the symptoms are those of an abundant hæmorrhage in one side of the cerebrum. On superficial examination it may appear remarkable that, even in large hæmatomata of one side, there is occasionally no

hemiplegia, or else it is very incomplete; but we must bear in mind that hæmatoma occurs just at the place where the increased pressure on one hemisphere is most readily transferred to the other, through the free communication between the two sides in the anterior portion of the skull, particularly when the hæmorrhage comes on slowly. Among the symptoms of hæmatoma, *Griesinger* also lays stress on the almost constant contraction of the pupil, and is inclined to regard this as a "symptom of irritation of the surface." In the previous chapter I attempted to give another explanation of the contraction of the pupil (which was also hypothetical) in diseases encroaching on the space above the tentorium.

Treatment.—In the treatment of meningeal hæmorrhages, the same rules hold good as were given for that of cerebral hæmorrhages, and we may the rather refer to the former chapter, as it is impossible to make an exact diagnosis between the two.

If we consider the diagnosis of a hæmatoma as certain, we may, in recent cases, apply leeches behind the ears, ice-compresses to the head, and give a purge occasionally. In the latter stages, blisters or pustulating ointments to the nape of the neck suffice. With this treatment I have had very good results in two cases that I have observed; still, in spite of the very characteristic symptoms in these cases, there may, possibly, have been an error of diagnosis.

INFLAMMATIONS OF THE BRAIN AND ITS MEMBRANES.

In the following chapters we shall speak—1. Of inflammation of the dura mater and its sinus. 2. Of inflammation of the pia mater, with puro-fibrinous exudation. 3. Of tuberculous inflammation of the pia mater, including acute hydrocephalus. 4. Of epidemic cerebro-spinal meningitis; and 5. Of inflammation of the brain-substance. Since inflammation of the arachnoid cannot be distinguished from that of the pia mater, we shall not treat separately of arachnitis.

CHAPTER VII.

INFLAMMATION OF THE DURA MATER, INFLAMMATION AND THROMBOSIS OF THE CEREBRAL SINUS.

In the previous chapter we spoke of one form of pachymeningitis, because, on account of the hæmorrhage it causes, and its clinical course, it is most suitably classed among meningeal hæmorrhages. Now we have only to speak of that form which, since *Virchow's* studies on hæmatoma of the dura mater, is called *pachymeningitis externa.*

Etiology.—It is very doubtful whether pachymeningitis occurs, as a primary and independent disease, from cold and other causes. At all events, it is generally secondary, and as such accompanies fissures, fractures, and especially caries of the cranial bones, particularly of the petrous and ethmoid bones, as well as caries of the upper cervical vertebra. In periostitis of the outer surface of the skull also, pachymeningitis occasionally occurs, without our being able to distinguish any continuity between the two diseases by changes in the cranium.

Inflammation of the cerebral sinus, with consequent thrombosis, or even thrombosis of the sinus, with subsequent inflammation of its walls, occurs proportionately often, being most frequent in the sinus lying on the petrous bone, the lateral and petrosal. This is evidently because inflammation and thrombosis of the cerebral sinuses are induced, in the great majority of cases, by caries of the petrous bone advancing to the base of the skull. The numerous patients suffering from tedious otorrhœa, as a result of otitis interna, are constantly threatened, as with a Damocles' sword, by inflammation or thrombosis of these sinuses. Not unfrequently there are suppuration and breaking down of the thrombus, and then particles of it may enter the efferent veins and lead to embolism and metastatic inflammation.

Anatomical Appearances.—The anatomical changes in mild and chronic cases of pachymeningitis externa are limited to a gradual thickening of the dura mater as a result of proliferation of connective tissue on its outer surface; the dura mater thus becomes firmly attached to the cranium, and subsequently the neoplastic tissue partly ossifies. In acute and severe cases the dura mater is usually reddened by vascular injection and small ecchymoses, is thickened and infiltrated in circumscribed spots corresponding to the point of injury or caries of the cranial bones. Later, this membrane becomes discolored, its tissue is relaxed and softened; finally, there is suppuration, and, if pus collects between the dura mater and cranium, the inflamed part becomes detached from the bone. In the latter case the pia mater also is almost always inflamed, and generally to a great extent. Even on autopsy it is often difficult to decide whether the inflammation of the wall of the sinus preceded the thrombosis, or the reverse. If the thromboses be not disintegrated, they adhere firmly to the relaxed rough inner surface of the thickened wall of the sinus, and occasionally extend backward to the torcular Herophili, and in rare cases down to the internal jugulars; as has been shown by the observations of *Lebert*, who has very greatly advanced our more accurate knowledge of this disease. More frequently on autopsy we find the thrombus already broken down, and the inflamed sinus filled with a purulent or sanious, occasionally gray-green and stinking fluid, mixed with flocculi. Along

with these changes we generally find those of otitis interna, and extensive caries of the petrous bone; viz., destruction of the drum, absence of the ossicula, polypoid proliferations of the mucous membrane, the tympanum full of pus, which also infiltrates the labyrinth, cochlea, and mastoid cells.

Symptoms and Course.—Chronic pachymeningitis, which causes thickening of the dura mater, and its firm adhesion to the cranium and ossification of the thickened layer, may be accompanied by headache and other symptoms; but these are not at all characteristic and do not show us what the disease is. The symptoms and course of acute pachymeningitis externa also are almost always so modified by the symptoms of the original disease, by complications with extensive inflammation of the pia mater, and by extension of the inflammation to the cerebral sinus with its results, that it is impossible to give a pure description of the disease. If an injury of the skull, or, still more, if a caries of the temporal bone, due to otorrhœa, be accompanied by unusually severe and extensive pain in the vicinity of the diseased bone, by fever, vomiting, dizziness, noises in the ear, twitching, delirium, and other symptoms of irritation of the brain, which are subsequently followed by those of depression, and finally by general paralysis, we may conclude that the disease of the bone has at first caused inflammation of the dura mater, and later, diffuse inflammation of the pia mater. The first stage is often short and indistinct, and, on the first visit or at their reception into the hospital, we find the patients in deep sopor. But even in such cases the above diagnosis may be made with tolerable certainty, if we find an injury of the skull, or particularly a chronic otorrhœa, and can discover no other causes for the brain-disease.

The symptoms induced by inflammation and thrombosis of the cerebral sinuses are always accompanied by those of meningitis as given above; hence it will be enough to limit ourselves to pointing out under what circumstances we are to suspect inflammation or thrombosis of a cerebral sinus, when we have a meningitis or encephalitis accompanying caries of the petrous bone. From the great frequency of this complication, I think it is well to keep in view its possibility and even its great probability, although the very doubtful signs of the disease should be absent. The suspicion will acquire more probability, should we observe a symptom of thrombosis of the transverse sinus that was given by *Gerhardt* with great acuteness, namely, less fulness of the jugular vein drawing its blood from the obstructed sinus. The same would be true if we found a symptom given by *Griesinger*, which was only observed in one case, a circumscribed painful œdema behind the ear; although, in caries of the mastoid process, this œdema

(which *Griesinger* calls a phlegmasia alba dolens en miniature) may arise from other causes than from the extension of the thrombus through the emissaria santorini which pass out in the sigmoid fossa. In most cases it is only from the occurrence of rigors and the signs of metastatic deposits in the lungs that we can conclude that caries of the petrous bone has not only induced meningitis and encephalitis, but has also led to thrombosis in the cerebral sinuses.

Treatment.—On the signs, or even suspicion, of inflammation of the dura mater, we should use energetic antiphlogistic treatment by repeated application of leeches behind the ears. At the same time, if there be any otorrhœa, we should make warm injections into the affected ear and cover it with cataplasms. Active purges and large blisters to the back of the neck are also useful. In other respects, the treatment of pachymeningitis corresponds with that of inflammation of the pia mater.

CHAPTER VIII.

INFLAMMATION OF THE PIA MATER, WITH PURO-FIBRINOUS EXUDATION—MENINGITIS OF THE CONVEXITY—MENINGITIS SIMPLEX.

Etiology.—In acute meningitis, an exudation containing many pus-cells is effused into the subarachnoid space; in chronic meningitis there are diffuse cloudiness and thickening of the pia mater and arachnoid, from proliferation of the connective tissue.

Acute meningitis, with puro-fibrinous exudation, is in many cases a secondary disease, and as such accompanies injuries and diseases of the skull and of the dura mater or other inflammations and other diseases of the brain. Except in the epidemic form, of which we shall speak in Chapter IX., it rarely occurs as an independent disease in previously healthy persons, but is somewhat more frequent in cachectic individuals or in those exhausted by long illness. Thus it is observed in convalescence from pneumonia and pleurisy, or from acute exanthemata and other infectious diseases, and from protracted diarrhœas, but especially during *Bright's* disease, etc. Although in these cases we frequently cannot discover any new source of injury acting on the body, we have no right to consider this inflammation of the pia mater as metastatic or even as secondary. The action of the sun's rays or of a very high or even of a very low temperature on the head, chilling the body, or getting wet, misuse of liquor, etc., are mentioned among the exciting causes of meningitis. But only the last of these causes has been proved to have any influence in exciting this disease. Recently *Griesinger* has called attention to a form of meningitis which

appears as one symptom of constitutional syphilis; and in the Greifswalder clinic I have seen a case of this kind, which has been fully described by Professor *Ziemssen*, at that time my assistant.

Anatomical Appearances.—Meningitis, with puro-fibrinous exudation, occurs chiefly on the convexity of the cerebrum. In the acute form we there find the small vessels of the pia mater more or less distinctly injected, and in the subarachnoid space, especially between the convolutions and around the large vessels, we find a yellowish, generally firm exudation, consisting of pus-corpuscles and fine granular fibrin. In milder grades of the disease the exudation is chiefly in the perivascular spaces. Occasionally the arachnoid is at the same time covered with a more fibrinous or more purulent coating. The cortical substance of the brain is sometimes of normal consistence, sometimes it is softened by inflammation. The ventricles which, in tubercular basilar meningitis, are almost always filled with fluid, are generally found empty in purulent meningitis of the convexity. In chronic meningitis we usually find the arachnoid adherent to the dura mater either by a few points or throughout a considerable extent; the pia mater is thickened and cloudy, the subarachnoid spaces filled with turbid fluid; or else we find the pia mater also transformed to a firm, decidedly thickened membrane, which cannot be removed from the brain without tearing.

Symptoms and Course.—Acute inflammation of the pia mater is accompanied by symptoms of severe fever, particularly by a very frequent pulse, and, like acute and extensive inflammation of other organs, occasionally begins with a chill. Fever of similar character and equal severity occurs in scarcely any other disease of the brain, and consequently is very important in the diagnosis of meningitis. If the frequency of the pulse disappear after the disease has lasted some time, if it fall from 120–140 beats per minute to 60–80 beats, while the other symptoms of fever and the functional disturbance of the brain increase, the evidence is still more in favor of meningitis. The other symptoms of the disease are headache and the repeatedly-mentioned functional disturbance of the brain, partly with the character of irritation, partly of depression or complete paralysis. In acute meningitis, the headache becomes very severe; patients not only complain of it while they retain consciousness, but even when this is impaired they frequently grasp the head and moan slightly, so that we may suppose they still feel pain. In almost all cases there are psychical disturbances even at the outset of the disease, probably from the vicinity of the cortical substance; the patients are very excited and restless, usually quite sleepless, and soon become delirious. In the sensory functions also there is more irritability, so that the patient

is very sensitive to light, sound, and even to slight friction on the skin. [A mere touch, especially upon the skin of the legs, will make the patient cry out; and even after the stage of stupor has set in he will wince if touched, showing that reflex irritability still continues. This hyperæsthesia of the extremities is a sign that the meningitis has extended to the spinal cord. Autopsies conducted by *Gerhardt*, both in children and in adults, uniformly support this interpretation of the symptom. On the other hand, the contraction of the neck-muscles so common in meningitis, which has also been ascribed to the coexistence of cerebral and spinal meningitis, is found more frequently in pure cerebral meningitis than when complicated with meningitis of the cord. In some cases a marked drawing-in of the belly affords a noticeable diagnostic mark.] Finally, there are noises in the ears, sparks before the eyes, restlessness, gnashing of the teeth, twitchings, and often also contraction of the pupil, and vomiting. We have had to mention all these symptoms in treating of simple hyperæmia of the brain and of hydrocephaloid; indeed, there is no pathognomonic sign of meningitis which is present in this and absent in other brain-diseases. It is true, the etiology, the grade of the fever, especially the height of the pulse and the unusual severity of the headache, occasionally speak, even in this stage, with great probability against simple hyperæmia or anæmia of the brain; but frequently it is only the subsequent course, the severe accidents that characterize it, the want of success in treatment, and the usually fatal result, that render the diagnosis certain.

If any of the characteristic symptoms be absent in the first stage, we must give a guarded diagnosis. Frequently only the effect of a laxative or of local blood-letting verifies the diagnosis between hyperæmia of the brain and meningitis. An attack of convulsions, usually preceded by stiffness of the neck, from tonic contraction of the muscles of the nape of the neck, often indicates the passage into the second stage. In this the patient falls into a deep sopor, becomes entirely insensitive to the irritation, and cannot move his limbs, although certain muscles, particularly those on the back of the neck, are in a state of tonic contraction, and there are general convulsions from time to time. [For some time after the commencement of the stage of stupor, periods of excitement recur, with delirium and loud screaming and convulsive paroxysms. It is remarkable that even at this stage of the malady fluctuations occur, with temporary improvement in the patient's condition; sometimes even the consciousness returns, and other deceptive signs inspire a hope of final recovery. The most important diagnostic marks of depression are the palsies,

which by their appearance often set at rest any remaining diagnostic doubts. These palsies are rarely complete hemiplegias, and seldom are they so marked as when arising from local disease of the brain ; but they ought not to escape the attention of any but a superficial observer. Sometimes an arm or a leg loses its strength. More commonly, however, there is a partial palsy of the face, such as dilatation of the pupil with insensibility to light, squinting, or the like.] The pulse also is usually, but not always, retarded. While the stupor and general paralysis increase, the patient generally dies of coma in a few days, more rarely not till the second or third week. These stages in the clinical course, between which there is occasionally a slight improvement, cannot be referred to any perceptible change in the pathologico-anatomical course ; as if, for instance, the first stage corresponded to the hyperæmia of the meninges, and the second to the exudation in the subarachnoid space. Nor must we omit to mention that not unfrequently, especially where the meningitis is due to caries of the petrous bone or to disease of the brain, the first stage is very little marked or not at all noticed. In these cases the symptoms begin with an attack of convulsions, which is repeated several times, and is followed by deep coma and general paralysis, usually accompanied by contraction of certain muscles.

[Stories of recovery from acute meningitis must be accepted with hesitation, error of diagnosis being a more probable event. Nevertheless, limited and mild inflammations of the pia mater undoubtedly have got well, but convalescence is very slow, and various derangements of the nervous apparatus persist for a long time. In chronic cases, and less often in acute ones, there may be imperfect recoveries, with permanent damage to the meninges and cortical matter of the brain—with trembling paresis, numbness of a limb, squinting, or deafness ; sometimes there are epileptic fits. The progressive palsy of the insane appears to depend upon a creeping inflammation of the meninges and upon periencephalitis, with occasional exacerbations and rises of temperature. The periods of mental excitement, headache, and convulsions correspond to those of elevation of temperature. Many other cases of insanity, characterized by psychical depression and ending in idiocy, must also be attributed to permanent injury to the cortical substance and meninges from inflammation.

The diagnosis of meningitis may present great difficulties. It has no pathognomonic symptom, and it is only by a careful grouping of the signs and a history of the case that a positive determination can be made.]

The symptoms of chronic meningitis are not accurately known, fre-

quently as its remains are found on autopsy of drunkards and insane patients. This is especially true of the commencement of the disease. It is probable that it is accompanied by headache and functional disturbance of the brain of an irritative character; but in drunkards, for instance, it will always be doubtful whether these are symptoms of inflammation of the brain or of alcoholic poisoning. The advanced stages of the disease may be more readily made out. If, in a patient who has been exposed to the above causes, and in whom we can exclude other brain-diseases, we find decided impairment of memory, dulness of intellect, disjointed frame of mind, if there be also trembling of the limbs, tottering gait, and other symptoms of gradually-progressing paralysis, we may diagnose chronic meningitis.

Treatment.—There is no doubt that favorable results are sometimes attained by active treatment in acute meningitis with puro-fibrinous exudation. It is not generally proper to bleed from the arm, but we may apply leeches to the brow and behind the ears, and, if the strength of the patient permit, may repeat the application. We may also cover the shaved head with cold compresses, and give an active purge of calomel and jalap. In the later stages of the disease, if, in spite of the previous treatment, there be coma and other signs of cerebral palsy, we may apply a large blister to the nape of the neck, and rub pustulating ointment on the head. Still more efficacious than these derivatives are douche baths, pouring cold water over the head from a pitcher held some distance above it. The patients almost always recover consciousness as this is being done; but, it must be repeated at intervals of a few hours, to secure a permanent result; with each successive employment of the douche we must increase the number of pitcherfuls. I shall lastly mention that frictions with mercurial ointment, and the continued administration of calomel, are much employed. In chronic meningitis, *Kruckenberg* also recommended cold douche baths as the most efficacious treatment. He particularly quoted the case of an old official, over whose head he daily poured as much as fifty pitcherfuls of cold water.

CHAPTER IX.

BASILAR MENINGITIS, TUBERCULOUS INFLAMMATION, AND SIMPLE MILIARY TUBERCULOSIS OF THE PIA MATER—ACUTE HYDROCEPHALUS.

Etiology.—In basilar meningitis there is a deposit, in the subarachnoid space at the base of the brain, of a readily-coagulating exudation, containing few pus-corpuscles. At the same time, there are almost always tubercular granulations in the meninges, causing a form

of disease designated as tuberculous basilar meningitis. This must be distinguished from simple miliary tuberculosis of the meninges, where the development of tubercle is not accompanied by inflammation and exudation, because the symptoms and course of the two diseases differ in some respects. Both basilar meningitis and simple tuberculosis of the meninges almost always cause effusions of fluid in the ventricles, and softening of their walls and the surrounding parts. The latter is the result of maceration, or destruction of the brain-substance by a simple transudation or inflammatory exudation.

Tuberculous basilar meningitis, as well as miliary tuberculosis of the meninges, rarely occurs, as a primary and independent disease, in persons previously healthy. We must bear this in mind, as it is very important for the differential diagnosis of the various forms of meningitis. These diseases are most frequently parts of a general acute or chronic miliary tuberculosis, or, at least, of one affecting most of the organs of the body. This form of the disease has many victims among those children of whom, when speaking of pulmonary tuberculosis, we said they had a strong predisposition to pulmonary consumption, if they did not die early of croup or hydrocephalus. These are not only children with marked scrofulous diseases, but also the offspring of consumptive or otherwise debilitated parents. They are badly nourished, and not well developed physically, but are often very bright mentally; have a fine skin, very perceptible veins, long eye-lashes, and a blue sclera. Caseous degeneration of the bronchial and mesenteric glands, caseous deposits in the lungs, and other old disturbances of nutrition, which, along with fresh deposits of tubercle in different organs, are found on autopsy of these children, if they finally die of hydrocephalus, cannot usually be recognized with certainty during life; hence the brain-affection is regarded as a primary disease. The case is different when the tuberculous inflammation or miliary tuberculosis of the meninges attacks adults. For then the symptoms of meningeal tuberculosis have either been preceded, for a short time, by those of acute miliary tuberculosis, or, more frequently, for a long time, by those of chronic consumption of the lungs. In other cases, tuberculous meningitis and miliary tuberculosis of the meninges accompany old tuberculous affections, such as chronic pulmonary or cerebral tuberculosis, and cheesy degeneration of the bronchial and mesenteric glands, but are not accompanied by fresh deposits of miliary tubercle in other organs; they form the only acute complication of these chronic tuberculous affections. Lastly, although rarely, tuberculous meningitis or miliary tuberculosis of the meninges occurs, without any precedent tuberculosis of other organs, in previously healthy persons, or during convalescence from severe diseases, such as typhus, measles, etc.

During the first year of life, tuberculosis of the meninges is rare; later, during childhood, it is proportionately frequent; in adults, only solitary cases occur, except where it complicates chronic pulmonary consumption. Among the exciting causes, premature or excessive mental exertion is blamed most frequently for exciting hydrocephalus in children; this is probably unjust. Children not predisposed to it may be mentally stimulated to any extent without inducing hydrocephalus; and the early development of children falling a prey to this disease is due to their predisposition, not to their bringing up; this is not the cause of their hydrocephalus. The same is true of the assertion that a blow or fall on the head induces tuberculosis of the meninges and acute hydrocephalus. It is almost always easy to make out that the sick child has, some time or other, fallen on its head; but it does not thence follow that this fall is to be regarded as the cause of his disease.

Anatomical Appearances.—In basilar meningitis we find a yellowish, opaque exudation, which is sometimes very plentiful, in the meshes between the pia mater and arachnoid, especially about the optic chiasm, as well as in the portions of the meninges extending toward the pons and medulla oblongata, and thence along the larger cerebral fissures, particularly the fossa of Silvius, toward the convex surface of the brain. At the same time, we almost always find the pia mater covered with whitish granulations, the size of a grain of sand or a hemp-seed, most distinctly so in the vicinity of the blood-vessels.

In simple miliary tuberculosis of the meninges the changes are less marked, and it is only on careful examination at the above places that we find numerous, usually very small, whitish, granular opacities of the pia mater, whose significance is often only rendered evident by the coincident occurrence of hydrocephalus and the presence of tubercle granulations in other organs.

In both forms, the ventricles, especially the lateral and the third ventricles, are sometimes moderately, sometimes considerably dilated by serous fluid. The latter is occasionally quite clear, but is usually clouded by flocculi. The walls of the ventricle, but especially of the fornix and commissures, are at the same time so softened that they usually break down on the slightest touch. This (hydrocephalic) softening spreads indefinitely, often far beyond the immediate neighborhood of the ventricle. The larger the effusion in the ventricles, the more bloodless and pale the brain becomes, and the whiter the softened parts.

Symptoms and Course.—There is no symptom pathognomonic of basilar meningitis, and which alone will render a diagnosis possible.

Nevertheless, the disease is almost always easy to recognize and to distinguish from other diseases. Characteristic peculiarities in the sequence of the symptoms, distinct signs from which the original seat of the affection and its subsequent extension may be determined, as well as the very regular subacute course of the disease, almost always give sufficient grounds for a certain diagnosis. At the commencement it is a local disease, situated at some point on the base of the brain, where numerous nerves start from that organ and run to the foramina through which they leave the skull. Later, when the disease extends to the ventricles, the local disease at the base becomes complicated, by the excessive effusion in the ventricles and by extensive hydrocephalic softening of the brain-substance, with diffuse disease of the cerebrum. In accordance with this, among the most constant symptoms of basilar meningitis are such as indicate irritation and afterward paralysis in the parts supplied by the nerves of the eye, the vagus and medulla oblongata. Among these are contraction and, later, dilatation of the pupil, ptosis of the upper eyelids, vomiting, slowness and subsequent frequence of the pulse, the peculiar changes of the respiration, and depression of the abdomen, according to the results of *Budge's* observations. In the same way, corresponding to the course of the pathologico-anatomical changes, the functions of the cerebrum at first show no marked disturbance, except the symptoms of so-called general cerebral irritation, while, as the disease advances, there is severe disturbance, of the character that we have frequently mentioned as due to compression of the capillaries by diseases encroaching on the space; such as loss of consciousness, epileptiform convulsions, paralysis of the extremities, etc. In cases where this second set of symptoms is not very marked, we may even conclude that the effusion in the ventricles is not very large. Lastly, when the symptoms of paralysis are limited to one side in certain cases, or are more decided on one side than on the other, it is usually because the hydrocephalic softening is more advanced on one side than on the other.

Although basilar meningitis, particularly its tuberculous form, and miliary tuberculosis of the meninges, very rarely occur in persons previously healthy, we must not count among its premonitory symptoms those of the diseases which it usually accompanies. On the other hand, in most cases, especially in children, the violent symptoms characteristic of a later stage of the disease are usually preceded for a time by insidious and indefinite symptoms, which may correctly be considered as premonitory. Such children show a change of manner, have no desire to play, like to sit in a corner, rest the head on the hands, are sleepy, and dream a great deal. They do not always complain of severe headache, and when they do it is especially in cases where there

s not simple miliary tuberculosis, but a tuberculous inflammation of the meninges. During these symptoms, which usually last for several weeks, the children become emaciated because their digestion is impaired, and probably also because they have fever. Nevertheless, it often happens that the mother overlooks or pays little attention to this period, and subsequently, when severe symptoms appear, assures the physician that the child was suddenly taken sick. We must know how important it is to decide whether there is the beginning of a new disease, or the commencement of the final stage of an old one, so as to make a more accurate examination. I have often found that the relatives and neighbors had noticed the altered manners of the child, while the mother had entirely overlooked them.

If the above symptoms be accompanied by vomiting, which cannot be traced to errors of diet, and does not occur after eating, but when the child is raised up, if the patient at the same time suffer from constipation and the abdomen is sunken, the physician must regard the case as very serious, although the parents usually think it free from danger. The little patients soon begin to complain more of their heads; they become sensitive to light and sound, gnash their teeth during sleep, and occasionally give a piercing cry (*crie hydrocephalique*). From time to time we see twitchings of certain limbs, or sudden spasm of the entire body. The patients start from their sleep with terror at some dream, and do not find relief from it even in waking. They are very much excited, and often repeat the same word or phrase innumerable times. At this time the pupils are usually contracted and the pulse is more frequent. When these symptoms succeed the premonitory stage, and when we at last see the child bending its head backward, boring into the pillows, with the muscles of its neck contracted, and the cervical lymphatic glands swollen, we become certain of the sorrowful fact.

After a few days, or even sooner, the scene usually changes very suddenly. The passage into the second stage is generally marked by an attack of general convulsions, such as we have already described. The vomiting then becomes rarer or ceases entirely, the children no longer complain of pain, but put their hands to their heads in a peculiar way; loud noise does not disturb them, and they do not turn the face away if a bright light be held before it; the peculiar cry and the gnashing of the teeth continue. We often find the muscles of some of the limbs or of one half the body slightly contracted, while those of the other side are relaxed. Now the previously-contracted pupils become dilated, occasionally first one, then the other; the children no longer regard objects held before them, and they begin to squint. The previously-frequent pulse becomes slower, falling to sixty beats a

minute or even less. The respiration generally shows very peculiar changes; for a time the inspirations are very superficial, and it almost looks as if the child forgot to breathe; then there is a deep sighing inspiration with which it repairs the neglect, as it were. The coma gradually becomes deeper; the lucid intervals that at first interrupted it grow more incomplete and shorter; the eyelids are either not closed, and the eye stares into space, or else the upper lid droops, and the eyeball is rolled up, so that the pupil is half covered by the upper lid. At the same time the color of the face often changes in a short time, and such a child, with its blooming cheeks, widely-open eyes, which have a peculiar dark and brilliant appearance from the dilatation of the pupil, has, to an uninitiated person, the appearance of not being very sick. During this stage there usually are attacks of convulsions at intervals; these are sometimes on one side of the body, sometimes on both. We must not conclude, from the convulsions on one side the body, that the opposite side of the brain is chiefly affected. The tetanic contractions of the muscles on the back of the neck, and the backward curvature of the neck, usually continue in this stage.

The stage of the disease last described occasionally continues a week or more. In spite of the hopelessness of the patient's state, the physician must continue his visits, and at each one he is asked anew if there is no chance of averting the danger; at last the parents are utterly overcome, but even then the disease occasionally continues for days, or a temporary appearance of improvement again excites deceitful hopes. These are hard times for the physician, especially as he is almost powerless against the slowly-advancing but inevitable result; and as, for days, he has but little to do except prepare the parents for the child's death. We must note that death is not imminent till some decided changes occur in the symptoms, to which it is well to call the attention of the attendants. Almost always twelve to twenty-four hours before death the pulse becomes very frequent, the skin is covered with copious perspiration, the previously-sunken belly becomes puffed up; defecation and urination are involuntary, and we may hear extensive moist and uneven *râles* in the chest.

Most cases of tuberculous basilar meningitis, and of miliary tuberculosis of the meninges in children, run their course in the above manner and resemble each other very much. Slight differences result from the predominance of certain symptoms, and from the different durations of certain stages or of the whole disease. But we must add that in some cases the disease runs a much more rapid course, and is accompanied by symptoms so similar to those of acute meningitis, that it is impossible to distinguish them. On the other hand, we must mention that occasionally, on autopsy of patients who have died of consump

tion, we find miliary tuberculosis of the meninges, and slight amounts of hydrocephalus, which were not indicated by any prominent symptoms during life. These cases seem to show that, in the description of the disease first given, the premonitory stage belonged to the commencement of the cerebral affection, and not to its complications or to the general tuberculosis. Lastly, we must point out that tuberculous basilar meningitis, and miliary tuberculosis of the meninges, developing during advanced pulmonary consumption in adults, have no distinct premonitory symptoms, but are first recognized from the occurrence of contractions of the muscles of the nape of the neck, coma, slow pulse, dilatation of the pupil, and the other symptoms of the fully-formed disease. Death is the most frequent termination. Well-proved cases of recovery are very rare; but, even excepting the cases where the diagnosis was somewhat uncertain, there is no doubt that it has occurred. Neither the ordinarily variable course of the disease, nor the surprising remissions that usually occur, should induce us to give a more favorable prognosis; only a continued improvement of all the symptoms dare excite the hope that the disease will take a favorable course.

Treatment.—Until within a short time, the treatment of tuberculous meningitis and acute hydrocephalus presented no difficulty to the physician. The inflammation of so important an organ naturally required the employment of all kinds of antiphlogistics. At the outset of the disease, venesections were ordered, calomel and flor. zinci were administered, and mercurial ointment was rubbed in the nape of the neck, then the occurrence of calomel stools, or the first signs of salivation, were anxiously awaited, for they were a guaranty for the efficacy of the treatment. In the later stages, absorption of the exudation was to be induced; hence the mercurial frictions were continued, and diuretics and active derivatives, even moxa on the shaved scalp, or pustulating ointments to it, were prescribed. If, in spite of all this, the paralysis continued to advance, an infusion of arnica and camphor was given. The more this disease was recognized as one symptom of extensive tuberculosis, the more antiphlogistic treatment was abandoned; then the opposite error was fallen into, either nothing was done (expectant treatment), or else all the efforts were directed to the cure of the tuberculous dyscrasia. The best mode of treatment of tuberculous meningitis and miliary tuberculosis of the meninges is as follows: At the commencement of the disease, especially when severe headache indicates tuberculous inflammation rather than simple miliary tuberculosis, we should apply leeches behind the ears. This is the more urgently to be advised, as in this stage an exact diagnosis of the different forms of meningitis is scarcely possible, and we do not certainly know whether the disease

is not at first a simple inflammation of the meninges, which is subsequently accompanied by the development of the tubercle, after repeated relapse of the inflammation. When the local abstraction of blood proves beneficial, it may be repeated during subsequent relapses. In these cases, at the onset of the disease, laxatives and ice compresses may also be used. Otherwise the treatment is the same as that for meningitis, with puro-fibrinous exudation. But the treatment must be entirely different when the inflammatory symptoms are less decided, when the headache is slight, and the disease drags on slowly. Then a single venesection is admissible, it is true, but it rarely has even a temporary effect, and its repetition is almost always injurious. On the strength of two successful cases, opposed, it is true, by a large number of unsuccessful ones, I recommend large doses of iodide of potassium, continued for a long time. In the cases above mentioned, where recovery took place under this treatment, there was a very extensive iodine eruption, and an iodine catarrh of the nose. These signs of iodism were absent in the unsuccessful cases. It cannot be denied that cold douches have a palliative effect, but I would advise against their use in marked cases of tuberculous basilar meningitis, and consequent acute hydrocephalus. During the affusion, the children almost always recover consciousness, but it is only for a short time. Moreover, when not successful, the operation is very painful, not for the children, but for the persons around. The latter feel very much pained that the child was worried at the last, when it could not be assisted. The same is true of the application of moxæ, and of frictions to the scalp with tartar-emetic ointment. *Hasse* recommends very small doses of morphia ($\frac{1}{24}$ of a grain) even in the early stages, as he has seen undoubtedly beneficial effects from it in some cases.

CHAPTER X.

EPIDEMIC CEREBRO-SPINAL MENINGITIS.

From the fatal epidemics of this disease which have occurred of late years in Germany, cerebro-spinal meningitis has acquired a great interest for German physicians, to whom it was previously almost exclusively known from the accounts of French observers. In 1865, I wrote a treatise on epidemic cerebro-spinal meningitis; this article was well received on many sides, but also caused some opposition, because, in spite of the small number of my observations, I had ventured to make various hypotheses concerning the nature of the disease and the indications of the symptoms. But, on careful examination of the extensive literature on the subject, of late years, I have found nothing

decidedly new, and have satisfied myself that my views had been generally received; so, in the present chapter, I shall chiefly follow my previous work.

Etiology.—We are unacquainted with the injurious power whose action induces this form of meningitis, and whose spread over greater or less extents of country excites more or less extensive epidemics of the disease. But we may regard it as very probable that epidemic cerebro-spinal meningitis does not depend on atmospheric or telluric influences, but is rather due to an infection of the body with a specific poison. It is true, the occasional frequent occurrence of a disease in regions generally free from it, and even the affection of several members of the same family, do not alone justify the conclusion that the disease depends on infection. But the passage of an epidemic from place to place, as is often seen in epidemic cerebro-spinal meningitis, is an important evidence of miasmatic extension. Apparently there is no transfer of the disease by contagion, although solitary instances are brought up to prove the infection of one person from another.

But cerebro-spinal meningitis does not, by any means, belong to that class of infectious diseases, of which we may take typhus as an example, and of which we shall hereafter speak in a separate section. The consideration of this disease as a peculiar form of typhus, which was formerly so common in France, has been entirely disproved during the late epidemic in Germany. I separate this affection from classes of infectious diseases to which the different forms of typhus belong, on the following grounds: In the latter, the severe constitutional symptoms, especially the fever, for the most part, depend immediately on the reception of the infecting material into the blood, and the anatomical changes in the organs, caused by the infection, are very peculiar; they are induced only by infection with the specific poison. In epidemic cerebro-spinal meningitis, on the contrary, the fever and all other symptoms depend solely on the local disease induced by the infection, and on its injurious effect on the body, just as they do in croupous pneumonia or in erysipelas; and the changes in the meninges of the brain and spine are just the same as those sometimes induced in other ways. This circumstance also induces me to treat of cerebro-spinal meningitis among the local diseases, in spite of its miasmatic origin.

Epidemics of this disease are more frequent in winter than in summer, and usually disappear as warm weather begins. But there are exceptions to this, which contrast very remarkably with most epidemic diseases. Among the different ages, childhood has the greatest quota of cases and deaths. Persons of middle age are often attacked also, while the aged are rarely affected. Unfavorable hygienic influences

of all kinds, among which are crowding of barracks, dwellings, etc., favor the outbreak of the disease. Persons exposed to these influences are in greater danger, at times of epidemics, than those who live under more favorable circumstances.

Anatomical Appearances.—In recent cases, the result of autopsy is almost always as follows: The subjects show no emaciation, protracted rigor mortis, or extensive hypostasis; there are often groups of dried herpes vesicles on the face and other parts of the body; the muscles are dark; in rare cases they are pale (*Ziemssen*).

The cranium contains much blood; in the longitudinal sinus there is abundant fluid, or softly-coagulated blood; the dura mater is more or less tense, and occasionally covered with small hæmorrhagic or pachymeningitic deposits. There is usually no effusion between the dura mater and arachnoid; in the subarachnoid space there is an exudation, which, both in extent and character, occupies about a medium position between the pure purulent exudation in meningitis of the convexity and the puro-serous exudation in basilar meningitis. In almost all cases, the convexity of the cerebrum and the base of the brain are affected at the same time, the latter usually the more severely. The exudation appears to be peculiarly plentiful about the chiasm, in the fossa Sylvii, at the base of the cerebellum, and in the fissures of the cerebrum. The nerves, from the base of the brain, are often entirely embedded in it. The brain itself is more or less vascular, almost always less resistant, even becoming pulpy in the vicinity of the ventricles; the latter generally contain small amounts of purulent fluid, rarely large quantities of clear serum.

The dura mater of the spine is more or less vascular, occasionally very tense, especially at the lower part. Between the dura mater and spinal arachnoid there is rarely a clear or purulent opaque fluid. The arachnoid usually presents no peculiarity except a decided opacity. There is more or less purulent fluid between it and the pia mater. The tissue of the pia mater is infiltrated with sero-purulent exudation. This infiltration extends very irregularly; in most cases the cervical portion is free; the process first begins in the dorsal region, thence extends toward the cauda equina, and is almost exclusively on the posterior surface. The purulently-infiltrated spots form irregular humps, broader in the middle, smaller at the ends, which are usually connected together by small striæ accompanying the larger vessels. Even where the pia mater does not contain this puro-serous infiltration, its tissue appears thickened and cloudy. The spinal medulla itself is more or less vascular, occasionally infiltrated and relaxed. In a case related by *Ziemssen*, the central canal was dilated and filled with purulent fluid.

Except some accidental complications, there are no particular anomalies of the other organs; we should especially note here that the spleen is almost always normal.

The *post-mortem* appearances, in protracted cases, are known only from a very few observations. In one such case I found the exudation thickened, and partly affected with caseous metamorphosis, as well as considerable fluid in the ventricles. The same appearances have been noted by other authors.

Symptoms and Course.—I must again repeat that the symptoms and course of epidemic cerebro-spinal meningitis may be fully explained by the changes in the meninges of the brain and spine, and that, on this account, the malady differs from most other infectious diseases. Every thing that has been advanced in opposition to this view may be refuted by the simple fact that, in genuine croupous pneumonia, which no one classes among the infectious diseases, certain symptoms, such as the frequent herpetic eruptions, albuminuria, etc., are just as difficult to explain, from the inflammatory changes in the lung and the copious exudation in the alveoli, as are some of the *occasional* symptoms of epidemic cerebro-spinal meningitis.

Only in rare cases is the outbreak of the disease preceded by a premonitory stage, characterized by slight headache and pain in the back. Usually the scene opens with an unexpected chill of variable duration, which is soon accompanied by severe headache, and in most cases by vomiting. The headache rapidly becomes very severe, the patient grows very restless, tosses about constantly, the pupils are contracted, the intellect remains clear. The pulse is 80–100, the bodily temperature moderate, the inspirations increase to 30–40 per minute. Even at the end of the first or second day, rarely later, we notice that the head is drawn backward. At this time there is often a herpetic eruption near the mouth, on the cheeks, eyelids, ears, and occasionally on the extremities. The complaints about severe headache continue; the pain extends from the head to the nape of the neck and the back. The restlessness becomes excessive, the ideas confused, the pupils remain contracted, the belly is sunken, and the bowels are constipated. The pulse and respiration become more frequent, occasionally the pulse is over 120 and the respiration over 40 per minute; the bodily temperature still remains proportionately low, or rises to 103° or over. The third or fourth day of the disease the tetanic contractions of the muscles of the neck and back become more evident, and are occasionally accompanied by trismus; there is excessive opisthotonus; consciousness is lost, but the patient still tosses about in bed, the pupils remain contracted, constipation continues, the belly is sunken, urine is passed involuntarily, or else the bladder becomes distend-

ed, and must be evacuated with the catheter. The now unconscious patient falls into deep stupor, the moaning respiration is accompanied by moist râles, and death occurs with the symptoms of acute œdema of the lungs.

In some particularly severe cases the above symptoms develop far more rapidly; consciousness is lost even during the first day, while severe tetanic spasms of muscles of the neck and back draw the head far backward. Death may occur even on the first or second day, when the disease is very malignant.

Lastly, in some few cases (of which there can be no doubt) the disease runs a still more rapid course, and causes death in a few hours, from general paralysis, occasionally even without the appearance of the most characteristic tetanic symptoms (*méningite foudroyante*).

But the disease does not by any means always terminate fatally when it has commenced and run its course for the first few days in the manner above described. As favorable signs, we usually first notice that there is less jactitation, and the mind becomes clearer, while the complaints of pain in the head and back and the tetanus of the cervical and dorsal muscles continue, or only slightly decrease. If the improvement progresses, all symptoms of the disease may disappear in a few days, and the patient begin his tedious convalescence.

Occasionally improvement begins, but does not continue, and the disease drags on. In such cases convalescence may not occur for weeks. Headache, contraction of the neck or opisthotonus continue. Paralysis of the motor and psychical functions causes a complicated series of symptoms, and the majority of such patients finally die of gradually-increasing marasmus.

Lastly, I must mention the occasionally intermittent course of the disease. I have only seen one such case. *Hirsch* distinguishes three forms of the intermittent course: in the first it only occurs in the first stage; one or more attacks of evident premonitory symptoms pass away, but another follows which is immediately succeeded by the outbreak of the disease. In the second form there is a sudden remission of the symptoms; these again grow worse the next day, and occasionally this alternation occurs several times, usually with a more or less regular quotidian type. In the third form, which is far the most frequent (to which my case belonged), perfect intermissions are seen during convalescence. The symptoms remaining after the disease, particularly headache and stiff neck, regularly increase very considerably for some time, usually with a quotidian type, while in the interval the patient feels quite comfortable.

To the above view of the symptoms and course of cerebro-spinal meningitis I will now add a short description of the individual symptoms,

both of those that I have already mentioned, and of others of which I have not yet spoken, as they are less constant. Severe headache occurs even in those cases which, as méningite foudroyante, terminate in death in a few hours. In the cases that run the usual course, as long as the patients retain consciousness they complain of headache, either spontaneously or on being questioned, and it also seems as if the restlessness, groaning, and complaints of the patients, after the intellect is clouded, were partly due to the headache. Lastly, during an epidemic of this disease, there are almost always a few cases where, without the disease actually occurring, persons complain of very severe headache continuing for several days without any other apparent cause. We may consider such cases as abortive forms.

Cervical and dorsal pains usually begin very early either with the headache or very soon after; they are generally increased by pressure on the spinal processes of the vertebræ; with rare exceptions, this pain is far greater when the patients make voluntary movements of the spine or on passive motion being made. If the disease be protracted, the dorsal pains and their increase by movements of the spine may continue for weeks.

Painful sensations in the extremities, unmistakably neuralgic in their nature, and caused by irritation of the posterior roots of the spinal column, are not constant symptoms; occasionally they only occur on motion of the spine.

Hyperæsthesia and Anæsthesia of the Skin.—Usually for the first days of the disease, and in some cases during its whole course, the patients are very sensitive to any rough handling; their restlessness, groaning, and complaints are increased when they are turned over in bed, occasionally even if they be percussed. Later, we often see no reaction even when the skin is greatly irritated; but in such cases, when the patients are in a state of stupor, there is cerebral anæsthesia. Far more rarely, there is peripheral anæsthesia, during which, while the patient is quite conscious, he feels irritation of the skin very little or not at all. This symptom apparently depends on loss of excitability of the posterior roots from inflammation.

Tetanic spasms of the cervical and dorsal muscles are only absent in some few cases of méningite foudroyante. At first the head is only slightly retracted, later it may form almost a right angle with the body. This position of the head and the addition of opisthotonos in the dorsal and lumbar regions usually render it impossible for the patient to lie on the back. If the tetanus attain a high grade, it almost always affects the respiration. Occasionally it disappears shortly before death; more frequently it continues more or less severe till death or convalescence.

Epileptiform convulsions are rare, which is very remarkable, considering the exudation is often widely spread over the convexity of the hemispheres.

Paralysis.—Usually there is no actual paralysis till death; but there are a few cases where hemiplegia or paraplegia, and quite a number where paralysis of the facial, oculomotor, or of the abducens, were observed. It is not at all difficult to explain these paralyses; on the contrary, it is almost wonderful they are not more frequent.

Psychical Disturbances.—At the commencement of the disease, the intellect is almost always unclouded; the patients answer questions correctly. But they soon become ill at ease and very restless. Then questions prove annoying, and they will only give short and incomplete answers. The incessant jactitation, which is scarcely interrupted by pauses of a few minutes, is very characteristic in the first stage of the disease. Subsequently most patients have delirium of variable intensity, which finally gives place to a soporose condition.

Disturbances in the Organs of Special Sense.—Patients not unfrequently become blind from keratitis, which is probably induced by incomplete closure of the eyelids, due to paresis of the orbicularis palpebrarum, or from exudative choroiditis and neuro-retinitis, probably a result of direct propagation of the purulent infiltration along the optic nerve from the cranium to the eye. Deafness is remarkably frequent, so that we are almost obliged to suppose that it has several causes, among which, however, the most important, doubtless, is the propagation of purulent infiltration along the auditory nerve to the internal ear.

Among the eruptions, groups of herpes vesicles in large numbers are very often seen, and more rarely erythema, roseola, urticaria, petechia, and sudamina. The frequency of the exanthemata, and particularly the occasional symmetry of their occurrence, have led to the supposition that they might depend on irritation of the cutaneous nerves, as *Bärensprung* has shown to be the case in herpes zoster from neuralgia.

According to *Ziemssen's* numerous and accurate observations, the *fever* has no regular course. Very few temperature curves resemble each other; sudden leaps and exacerbations of short duration often occur. But generally a remitting type, with exacerbations of half a degree to a degree, is most frequent. Very high temperatures are almost exclusively seen in severe cases that terminate fatally. In most cases the temperature does not rise above 103°. The intermittent fever that occasionally accompanies the other symptoms during convalescence is regarded by *Ziemssen* as a *reabsorbing* fever, while he refers that occurring during the first and second weeks to an interrupted progress of the meningitis. The frequency of the pulse does not at

all accord with the height of the fever; with moderate fever it is occasionally very high; slowness of the pulse is only rarely observed at the commencement of the fever.

Treatment.—Just as in other malignant and fatal epidemics, in deciding the best means of treatment, we must only consider cases where there is at least a slight hope of recovery. Whoever tries any proposed plan only on the severest cases will attain negative results by any procedure. The customary treatment of sporadic meningitis, consisting of the energetic use of cold as ice-compresses to the head, the application of leeches behind the ears, and the internal administration of calomel, is also advisable in epidemic cerebro-spinal meningitis, as is very evident from its excellent effect in patients attacked by the premonitory symptoms of the disease, severe headache, and pain in the neck, during an epidemic.

But, according to most observers, this mode of treatment has preserved its reputation even in marked cases of the disease; there is but little opposition to it, and even this is based on its want of success in the severest forms. I have no personal knowledge of the success of morphium when given internally or hypodermically. But a number of trustworthy observers, *Ziemssen* and *Mannkopff* among others, speak most favorably of it, especially in the form of subcutaneous injection. *Ziemssen* says: "Although we have used morphia frequently, we have never seen any injurious effects from it, but, on the contrary, such a decidedly palliative action that, along with cold, it seems the most indispensable remedy in the treatment of meningitis." Almost all observers agree that quinine is entirely useless even in decidedly intermittent cases.

CHAPTER XI.

INFLAMMATION OF THE BRAIN—ENCEPHALITIS.

Etiology.—In encephalitis, just as in the inflammation of other organs containing little connective tissue, there is not much interstitial exudation, but there are most important changes in the nerve-filaments and ganglion-cells and their scanty interstitial tissue. These tissue-elements swell from absorption of nutrient fluid and subsequently break down, partly to simple detritus, partly after precedent fatty degeneration; in the latter case, in the inflamed parts we find quantities of fatty granular cells (the formerly so-called *Gluge's* inflammation globules), which we have a right to suppose come directly from fatty degeneration of ganglion-cells and neuroglia nuclei. In the subsequent course of encephalitis there is often an extensive formation of

pus-cells; abscesses are formed, which, besides the usual constituents of pus, often contain some remains of disintegrated brain-substance. Cerebral abscesses are either surrounded by brain-substance softened by extension of inflammation to the surrounding parts, or by newly-formed connective tissue; in the latter case they are said to be capsulated.

Encephalitis is a rare disease, and is not induced by the causes which usually excite inflammation in other organs. 1. The most frequent form is traumatic encephalitis; it results not only from direct injuries affecting the brain after the skull is opened, but there are numerous cases where the cranium has remained uninjured, and where there was apparently only slight contusion. It is most probable that in such cases the brain has been bruised by the vibrating cranium, and some small vessels have been ruptured and ecchymoses formed. It also appears as if the small extravasations of blood occasionally induce no symptoms at first, but excite inflammation in the surrounding parts, and thus subsequently cause encephalitis. At least, the first signs of the inflammation are occasionally not observed till long after the injury. Among the cases of traumatic origin we must include those where breaking down of the brain by an extensive extravasation of blood has caused inflammation in the vicinity. 2. In other cases the encephalitis depends on the irritation of the brain from neoplasia and necrosed spots. 3. Among the most frequent causes of encephalitis are diseases of the cranial bones, especially of the petrous bone. As we shall hereafter show, the presence of an otorrhœa may decide the question between an abscess of the brain and a tumor. Cases are also known where caries of a superior maxillary extended to the brain and caused encephalitis. I remember a clergyman in Magdeburg who died of abscess of the brain a few years after a large portion of the upper jaw had been exsected on account of extensive caries. 4. Occasionally the disease occurs in the course of acute and chronic infectious diseases, such as pyæmia, glanders, typhus, etc., without our being able to give any plausible explanation for it. 5. Lastly, but rarely, encephalitis occurs without any known cause in persons previously healthy.

Anatomical Appearances.—This disease never attacks the entire brain; it is always confined to certain points. The size of these spots varies from that of a bean to that of a fist, or larger. They are usually of an irregular spherical shape. Ordinarily there is only one, but sometimes there are several. They may be located either in the cerebrum or cerebellum; they are most frequently in the gray substance and very near the surface, if they do not quite reach it. In the commencement of the disease at the affected parts, we find the brain-substance swollen, infiltrated, softened, and pointed with red spots from

small extravasations of blood. The swelling of the inflamed parts may be recognized by the surface of the brain on the affected side appearing smooth, just as in apoplectic effusions, and the brain itself being anæmic from encroachment on the cranial cavity. On incision also the diseased portion not unfrequently rises above the level of the incised surface. After long existence, the relaxation of tissue gets the upper hand, and a red pulp results, which gradually acquires a rusty-brown or yellow color, from change of the hæmatin, or when there is a slight admixture of blood it becomes grayish. This pulp, which may be washed off by a slight stream of water, consists of remains of nerve-filaments, blood-corpuscles, granular cells, and fine granular exudations or masses of detritus. The subsequent changes in the inflamed parts, which are usually called the results of inflammation, vary. Occasionally, in the vicinity of the inflammation, there is a new formation of connective tissue, which is also continued through the inflamed spot as a delicate network; the contents of the abscess are absorbed, and there remains a cavity, filled with a chalky milky fluid, the "cellular infiltration" of *Durand-Fardel*, previously mentioned in apoplexy. In some cases, especially when these cysts lie near the surface of the brain, their walls gradually approach each other, while the contents disappear, and in place of the abscess there is formed a cicatrix, at first pale red and vascular, subsequently white and callous. When encephalitis terminates in suppuration, the appearances are different. Recent abscesses of the brain form irregular round cavities filled with yellow or gray, occasionally also reddish, thick fluid; their walls consist of ragged masses infiltrated with pus. In the immediate vicinity of the abscess we usually find inflammatory softening; farther off there is œdema of the brain-substance. Such abscesses increase till they break into a ventricle, or reach the meninges; or, if extensive meningitis do not occur in the latter case, the ulceration extends to the cerebral membranes and the skull, and finally the pus may perforate outwardly or into neighboring cavities, especially into the cavity of the tympanum. If the cerebral abscess be capsulated by new formations of connective tissue in its wall, it has a more regular form and smooth walls. If it has lasted a long time, we occasionally find the capsule much denser, and the contents thickened by reabsorption of the fluid part, and changed to a cheesy chalky mass.

Symptoms and Course.—Like the symptoms of cerebral hæmorrhage and of partial necrosis, those of encephalitis are partly the immediate result of destruction of the portion of brain affected, and partly the result of disturbances of circulation in the brain, especially in the vicinity of the seat of inflammation.

Since large portions of the brain may be destroyed without caus-

ing perceptible functional disturbances, and since encapsulated abscesses frequently do not decidedly affect the circulation in the skull, we may readily understand that we should occasionally find abscesses of the brain on autopsy, which had not been suspected during life. With this knowledge we may also understand those cases of cerebral abscess which run their course without any symptoms except the injurious influence they exercise on the general nutrition of the brain. It is not at all rare for a patient to have dull headache, increasing apathy, loss of thinking-power, even advancing to idiocy, a blunting of the senses, increasing weakness, and uncertainty of movement-symptoms which indicate the existence of severe brain-disease, it is true, but which do not by any means justify a diagnosis of abscess of the brain. Even the most experienced and accustomed observers occasionally mistake abscesses of the brain. But these and similar mistakes, which unfortunately are made public far less frequently than brilliant diagnoses, can only seem strange to the ignorant, and to those who are not thoroughly acquainted with the physiology and pathology of the brain, and with the diagnosis of brain-diseases.

These latent cases of encephalitis are far less frequent than those where the disease may be very strongly suspected or recognized with certainty. If the abscess be located at a point where it destroys the centres of special sense, or interrupts the conduction of the impulse of the will to the motor nerves, or the impressions of special sense to the organs, we have partial anæsthesia and partial paralysis, sometimes of the cerebral nerves, again of the cerebro-spinal, at others of both. These anæsthesias and paralyses not unfrequently extend to both extremities of one side of the body; in other cases they are confined to the parts supplied by certain cerebral or cerebro-spinal nerves, but in the latter case also they are always on one side. Bilateral anæsthesias and paralyses only occur exceptionally, and in cases where the abscess is in the middle portion of the brain, which is not double, or where different abscesses are in the same parts of the brain on opposite sides. From the occurrence of these symptoms we can only determine that there is some local disease; but we cannot decide on its nature, for they occur in the most varied forms of local disease where nerve-filaments and ganglion-cells are destroyed. To come to a conclusion on this point, we must pay particular attention to the etiology, to the course of the disease, and to those symptoms which show the effect of the local disease on the rest of the brain.

If we can make out that the symptoms of a local disease of the brain have begun after an injury of the head, or if the patient has caries of the petrous bone, the case is most probably one of encephalitis. If, on the other hand, there has been no injury of the head,

and no caries of the temporal bone can be found, it is almost equally improbable that there is encephalitis. While these points are generally true, they have in some cases led to erroneous diagnosis. Autopsy has revealed abscesses of the brain where there had been no injury of the head or caries of the cranial bones. There have also been cases where patients with cerebral tumors have had a blow on the head, and occasionally the previously latent brain-disease had not become manifest till after this injury. (These cases are analogous to those where women first have their attention directed to a carcinomatous mamma by a blow on it, and then consider it beyond a doubt that it is due to the injury they have received.) But, practically, it is best only to think of these exceptional cases where there is sufficient reason.

Encephalitis never affects the entire brain, but is always limited to certain points; nevertheless, at the commencement of the disease, there are almost always signs of temporary irritation of the entire brain or meninges, such as increased frequency or great slowness of the pulse, increased bodily temperature, headache, dizziness, sleeplessness or restlessness, exciting dreams, psychical irritation, even mild delirium, great sensitiveness to slight irritation of the organs of special sense, bodily disquiet, and great weakness, etc. According to *Griesinger*, this violent commencing stage, which may be followed by quiet, is almost pathognomonic of encephalitis and abscess of the brain. In the criminal records there are numerous cases where the above symptoms were considered by the attending surgeon as traumatic fever, because they apparently disappeared without any traces, and the complete recovery of the patient from the injury received was officially certified to, while, after a time, there were unmistakable signs of a severe brain-disease, and autopsy showed abscess of the brain as the fatal termination of the injury. Even in those cases where an injury of the skull is followed at first by the symptoms of general irritation of the brain and meninges, and subsequently by the above-described decrease of all the cerebral functions, we should first think of abscess of the brain, and attribute the absence of local symptoms to the location of the abscess at a point where it does not interrupt the conduction of excitement, in the centripetal and centrifugal cerebral filaments.

The influence that local inflammations in the brain have on the rest of the organ consists partly in their encroachment on the cranial cavity, and chiefly on those chambers of the skull where they are located, partly on the collateral hyperæmia and œdema in their vicinity, and, lastly, partly on their injurious effect on the nutrition of the entire brain. On the first of these factors depend the headache, the attacks of dizziness and vomiting, as well as the evanescent occurrence of pain,

indefinite feelings, anæsthesia, twitchings, contractions, paralyses in some parts of the body, which, in many cases, accompany the symptoms previously mentioned, and which we fully described in the second and fourth chapters, as the results of partial hyperæmia and partial anæmia of the brain. These do not continue unchanged, like the local symptoms, which are the immediate results of partial destruction of the brain from the inflammatory process, but are rather apt to have a variable course; this is partly due to occasional enlargement of the abscess, by which the space is more contracted, partly on the occasional increase and decrease of collateral hyperæmia and collateral œdema. It is evident that such a variation in the symptoms would occur oftener in encephalitic inflammations than in slow and steadily-growing tumors of the brain.

The attacks of epileptiform convulsions, which not unfrequently occur during encephalitis, are more difficult to explain, and we shall not even offer a hypothesis as to the mode of their occurrence. Statistics have been made as to how often headache, contractions, epileptiform convulsions, etc., were present or absent in a large number of cases of cerebral abscesses. In individual cases, the results of these statistics are, of course, of but little value in the diagnosis of abscess of the brain, or in its differential diagnosis from tumor of the brain. The injurious influence of the local inflammation on the general nutrition of the brain, which may occur earlier or later, but never fails when the disease is protracted, evinces itself by the previously-described symptoms of a general paralysis of all the cerebral functions. I think I have fully shown, by this description, that, in many cases, it is possible to recognize an abscess of the brain, although it has no constant or pathognomonic symptom.

The duration of encephalitis varies; some cases terminate in death, after a few days or weeks, while in others this does not occur for years. Occasionally, while the symptoms of cerebral paralysis increase, so that the patient becomes idiotic, we find a remarkable development of fat. Death occurs either suddenly and unexpectedly, from the development of meningitis, from sudden increase of the abscess, and, occasionally, without our being able to find, on autopsy, any cause for the sudden catastrophe; or else it occurs with the symptoms of gradually-increasing sleepiness, finally becoming deep coma, or else it is induced by intercurrent diseases. Recovery is very rare. Even in the most favorable cases, where the cellular infiltration forms a cicatrix or encapsulates the abscess, while its contents calcify, we can only speak of a relative cure, since both the remains of the inflammation and the atrophy of the brain, which usually develops under such circumstances, affect the functions of the brain for the rest of life.

Treatment.—The treatment of encephalitis cannot be very successful, from the simple fact that the disease is not generally recognized until it has induced irreparable destruction of the brain. In very recent cases, particularly in those of traumatic origin, we should employ local antiphlogistics, leeches, and cold compresses. Later, but little can be expected from this treatment. Moxæ and setons, which were formerly much used in protracted cases, have been abandoned, and very justly so, as has the administration of mercurials. The preparations of iodine also, which have been recently recommended, promise but little benefit; hence, in most cases, there is little to do but have the patient observe proper regimen, particularly to guard him from every thing that can increase the pressure of blood to the head, and to confine ourselves to combating the most urgent symptoms. Among the latter, the apoplectiform attacks are to be treated according to the rules given for the treatment of cerebral hæmorrhage, while any intercurrent meningitis demands the employment of energetic antiphlogistic remedies.

CHAPTER XII.

PARTIAL SCLEROSIS OF THE BRAIN.

In previous chapters we have repeatedly mentioned the formation of indurations in the brain as the results of cicatrization of apoplectic and inflammatory deposits. Besides these, partial scleroses, which occur as a result of other processes, indurations from new formations of connective tissue, and displacement of the normal elements, occur at circumscribed spots in the brain, whose pathogeny is entirely obscure, and of which it is even doubtful whether they are of inflammatory origin or not. These idiopathic forms of partial sclerosis of the brain have been almost solely observed early in life, and more frequently in males than in females, but their causes have never been discovered.

On anatomical examination the points of disease are found oftener in the white than in the gray substance; their number varies; sometimes they are solitary, occasionally several are present. They form irregular nodules, or rough spots as large as a lentil or an almond, which may be distinguished from the parts around by their hardness and toughness. On the cut surface they appear bloodless, dull, and milk-white; after remaining exposed for a while, they are covered with a scanty serum, and become somewhat depressed. In the midst of the nodules there are usually small blue or grayish-red spots, which contract strongly and exude a quantity of serum, after being

divided. On microscopical examination we find amorphous fine granular masses along with well-preserved nerve-elements in the milk-white indurated spots. In the grayish-red prominences, on the other hand, there are no nerve-elements; they consist of a filamentary mass and the remains of capillary walls, in which fat granules are embedded (*Valentiner*).

The symptoms and course of partial sclerosis of the brain are not so characteristic as to render the disease easy of recognition; on the contrary, it must be regarded as a triumph in diagnosis, that this has been done in a few cases (*Frerichs*). Since the sclerosis is limited to scattered and circumscribed points and develops slowly, the paralyses accompanying the disease have the peculiarity that, in the commencement at least, they do not extend over one-half the body, but always begin in single groups of muscles, or in a single extremity (always one of the lower extremities), and thence extend gradually to other groups of muscles and other extremities, till finally the central parts governing respiration, deglutition, and the actions of the heart, are affected. This extension is not regular: for example, paralysis of the right hand does not necessarily follow that of the right foot, but it is entirely lawless. This fact in itself speaks in any case for the dependence of the paralysis on numerous separate points of disease, and not on one gradually-increasing spot. Besides the paralyses, which are the most constant, and, from their peculiar mode of extension, the most characteristic symptoms of partial sclerosis of the brain, there are also disturbances in the other functions, but these are far less distinctive and less characteristic of the disease. Headache is absent in most cases; but there are often peripheral pains, and a feeling of formication in the extremities which is followed by a diminution of sensation, increasing to perfect anæsthesia. The special senses are rarely affected, and among these that of sight almost exclusively. In most cases there are moderate symptoms of irritation in the psychical functions; but these are soon followed by a gradually-increasing deep depression, which in some cases exists from the first. Convulsions only occur exceptionally; but trembling is a very constant symptom. The nutrition of the body is not generally affected till late in the disease, and in some patients, just as in encephalitis, there is even a remarkable development of fat. The course of the disease is very tedious; most of the cases observed lasted from five to ten years. During this time the disease occasionally appeared to remain at a stand-still, but no existing paralysis was ever observed to disappear. No instances of recovery are known. In the cases that have been described, death resulted partly from the extension of the disease to the medulla oblongata and consequent disturbance of the respiration, partly from

marasmus and hydræmia which developed toward the end, partly from intercurrent diseases.

Treatment is fruitless against this affection; it can only be of use in combating the more severe symptoms.

CHAPTER XIII.

TUMORS OF THE BRAIN AND ITS MEMBRANES.

Under the head of tumors of the brain are included growths and parasites occurring in the skull, and aneurisms of the cerebral arteries. We shall follow this custom, since it is very convenient on account of the great correspondence of the symptoms due to these otherwise different diseases.

Etiology.—The pathogeny and etiology of cerebral tumors is very obscure. This is true not only of carcinoma, sarcoma, glioma, and myxoma, but also of the rarer tumors, cholesteatoma or pearl tumors, lipoma, and cystoid growths. Cancer of the brain occurs chiefly in advanced age; it is sometimes primary, and then usually remains the only carcinomatous disease in the body; sometimes it is secondary to carcinoma of other organs. The other neoplasia that we mentioned above, also occur chiefly, but not exclusively, in advanced age, and according to statistics are more frequent in males than in females. We know nothing of the morbid predisposition or the exciting causes to which these neoplasia are due. They have been frequently observed after precedent injuries of the head; but in such cases there is no certainty of a genetic connection between the injury and the growth. We must bear in mind how frequently slight injuries of the head occur, and how carefully they are inquired after, in any patient suffering from symptoms of brain-disease, and, when found, how strong the inclination is at once to refer the disease to them.

Tubercles in the brain with rare exceptions occur in children, and chiefly in those over two years old. They are rarely primary; but almost always form complications of the so-called tuberculosis of the lymphatic glands, and of pulmonary tuberculosis.

Cysticerci and echinococci of the brain, as of other organs, depend on the emigration of the embryos of these parasites.

Aneurisms of the cerebral arteries are usually due to degeneration of the arterial walls from endarteritis deformans.

Anatomical Appearances.—Carcinoma of the brain is usually in the form of round or lobulated, generally circumscribed tumors, with the softness and other peculiarities of medullary cancer; more rarely they have the firm tissue of scirrhus, and then they usually pass into the neighboring cerebral substance without having any distinct bound

aries. They sometimes start from the brain itself, sometimes from the dura mater and cranial bones, or they develop originally from the external soft parts of the skull and neighboring cavities, especially in the orbit, and thence press into the skull. On the other hand, it rarely happens that carcinoma occurring in the brain perforates the meninges and cranial bones. There is usually only one carcinomatous tumor of the brain, and this is generally located in the cerebrum; where there have been several, they have occasionally been found symmetrically located on the two sides of the brain. Cerebral carcinomata, which may attain the size of the fist, never suppurate unless they perforate outwardly; on the other hand, they readily undergo partial retrogressive metamorphosis, become yellow and cheesy in the middle, shrink, and thus cause umbilicated depressions on the surface of the brain, if the cancer had advanced so far.

Sarcomata occur as often as carcinomata in the brain. They are often attached to the meninges, and the tumors of this variety, at the base of the brain, usually attain greater size than those of the dura mater that covers the convexity. (Just as often sarcomata are embedded in the midst of the brain-substance, in the majority of cases in the cerebrum.) They form round or lobulated tumors, from the size of a hazel-nut to that of a good-sized apple; their cut surface is smooth, dirty white, or grayish red; they are usually soft, even medullary, more rarely hard and fibrous. Occasionally they contain cavities filled with fluid. Sarcomata consist chiefly of spindle-shaped cells, arranged in filamentary striæ. They differ from cancer, and especially from glioma, not only in being sharply bounded, but in being often surrounded by a vascular envelope, from which they can be turned out. Small lumps of carbonate of lime not unfrequently occur in sarcoma of the dura mater; on rubbing the tumor between the fingers, these lumps cause a sandy feel. *Virchow* has designated tumors, containing numbers of the chalky lumps, as *psammonea*, or sand-tumors. Their mode of origin is not yet exactly known.

Myxomata consist of mucous tissue—that is, of variously-formed cells, embedded in a homogeneous, mucous, hyaline, intercellular substance. They are not very rare in the brain. Like sarcomata, they are located most frequently, but not exclusively, in the medullary substance of the cerebrum, where they generally appear as circumscribed tumors, more rarely as infiltrations of soft gelatinous substance. The tissue of myxoma is somewhat translucent, and of a weak yellowish or red color, but may acquire a varied hue from extravasated blood. Myxomata also correspond with sarcomata in regard to the size they may attain; and, between these two forms of tumors, there are all possible grades of transformations (gelatinous sarcoma).

Gliomata result from a local proliferation of the neuroglia, or connective tissue of the brain, at the expense of its nervous elements. Microscopically they consist of roundish nuclei, distributed through a finely-reticulated basement substance. By the naked eye they are distinguishable from not forming circumscribed tumors, but passing gradually into the healthy brain-tissue; also from the fact that they never pass from the brain to its membranes. Gliomata may attain the size of a fist; they most frequently originate from the medullary substance of the cerebrum. Hæmorrhages into or partial retrogression of glioma may take place after precedent fatty degeneration of its elements. The consistence of these tumors varies between that of a medullary cancer and of healthy brain. The cut surface varies in color from whitish yellow to bright grayish red, and usually shows a number of cut vessels. Formerly glioma was regarded as infiltrated cancer, and, as it occurs chiefly in young persons, or, at least, in those under forty years of age, we see why it was said that cancer of the brain has been observed at all ages.

Cholesteatomata, or pearl tumors, are rare. They sometimes start from the cranial bones, at others from the meninges, again they develop in the brain itself. They form irregular tumors, of variable size, of a pearly lustre; they are enclosed by a delicate membrane, and consist of concentric layers of epidermic cells.

Lipomata, as small lobulated tumors, starting from the dura mater, *cysts* filled with fluid, or fat and hairs, and *cystoid neoplasia*, with cauliflower excrescences from the walls, belong to the rarities, and have more pathologico-anatomical than clinical interest.

Tubercles of the brain are the most frequent form of cerebral tumors. Usually we find only one collection of tubercles, occasionally two, more rarely a greater number. The size varies from that of a hemp-seed to that of a cherry, or, in rare cases, to that of a hen's egg. They are located most frequently in the cerebellum or cerebrum, more rarely in the mesencephalon. Tubercles of the brain form irregular, roundish, non-vascular tumors, of a yellow, dry, and cheesy character. They are sometimes surrounded by a layer of loose connective tissue, which separates them from neighboring parts of the brain; sometimes the main portion of the yellow nodule gradually passes into the brain-substance as a gray-white, slightly-translucent, narrow margin, consisting of young tubercle elements. In the latter case the tubercle has been growing till death; in the former, where it can be readily turned out of its capsule, growth has ceased long before death. From softening of its centre, the tubercle nodule is occasionally transformed into a vomica, filled with tubercle pus.

Syphilomata only rarely occur in the brain in the form of nodules

(gummata); they are more frequent as diffuse infiltrations. Nodular syphiloma always passes from its edges very gradually into the healthy brain-substance. Since such nodules become changed by atrophy, and fatty metamorphosis of the cells commencing in the centre, to a substance resembling yellow tubercle, syphiloma has probably been occasionally mistaken for tubercle. This may be avoided by bearing in mind that, in syphiloma, the passage from the cheesy centre to the broad, grayish-white, peripheral zone, is very gradual, while, in infiltrated, growing tubercle, these zones follow each other more closely and, in tubercles that can be turned out, they do not exist. In regard to consistence, and the character of the cut surface, syphilitic infiltrations greatly resemble old, simply sclerosed parts; indeed, even the microscope cannot always decide between them.

Cysticerci are not very rare in the brain, and, when found, they are usually in large numbers; they generally occur in the gray substance. Occasionally some of these parasites are found dead, and changed to a mortar-like concrement, in which some of the hooks from the circle can usually be recognized.

Echinococci of the brain are much rarer. They form large vesicles, enclosed by a very delicate adventitia, with the well-known peculiarities of which we have often spoken.

Aneurisms of the cerebral arteries are not frequent. They chiefly occur in the vessels at the base of the brain, in the arteria basilaris, a. corporis callosi, a. fossæ Sylvii, and in the a. communicantes of the circle of Willis. In rare cases, they attain the size of a small hen's egg, while usually they only reach that of a pea or a small hazel-nut.

Symptoms and Course.—The symptoms of cerebral tumors have the greatest resemblance with those of the local diseases of the brain previously described. There is no symptom, occurring during the course of a cerebral tumor, that does not sometimes occur from softening of the brain, from abscess, or from some other local disease. This resemblance cannot astonish us; on the contrary, we could not understand how it should be otherwise, since, like other local diseases, a tumor of the brain destroys a circumscribed portion of the organ, encroaches on the intracranial space, and interferes with the circulation in the vicinity of the diseased part. Nevertheless, it is only in rare cases that it is difficult or impossible to recognize a cerebral tumor, and to distinguish it from an abscess, or other local disease of the brain. (According to my experience during the last ten years, I must hesitate about agreeing with the first part, at least, of *Bamberger's* assertion, which I have previously quoted, where he says, "With few exceptions, the diagnosis of tumors of the brain is rather a guess than a diagnosis.

and the determination of its locality, likewise with certain exceptions, is impossible.") This apparent paradox is due to the fact that, in spite of the absence of constant symptoms pathognomonic of tumors of the brain, in most cases the diagnosis may be made partly from the etiology, partly from the location of the disease at a point where other local diseases are rare and tumors common, and, lastly, partly from the peculiarities of the entire course of the disease.

The important evidence furnished by the etiology is chiefly negative. In every case of brain-disease we should make it a rule to seek for the exciting cause by obtaining an exact history of the case. If we thus find that the patient has had no injury of the head, that he has no caries of the petrous bone, no hypertrophy or valvular disease of the heart, and probably also no degeneration of the arteries; in short, if we can find no cause for the disease, our suspicions must first turn to a tumor of the brain. Before speaking of the cases where cerebral tumors can be recognized with facility and certainty, I shall say a few words concerning the rare cases where they induce no symptoms, or else have those of severe brain-disease, but do not give any means of determining that they are caused by a tumor. The tumors with latent course can only be located at parts of the brain where they do not disturb the intercerebral centres of the cranial nerves, or interrupt the conduction of excitement in the centripetal and centrifugal brain-filaments, that is, chiefly in the extensive medullary masses of the cerebrum. At this part tumors often attain a large size without inducing local symptoms (herdsymptome), while at most parts of the base of the brain, and in the vicinity of the large ganglia, even the smallest tumors are accompanied by these symptoms. But even in the cerebrum only those tumors run a latent course which grow slowly, and are not so vascular as to swell at times from the vessels being overfilled, and at others to decrease in size from containing less blood. To explain the remarkable but very certain fact that the symptoms accompanying rapidly-growing tumors are not unfrequently absent in those that grow slowly, a certain "power of accommodation" has been ascribed to the brain, which enabled it to accustom itself to gradually-increased pressure. I consider the following explanation more satisfactory: In tumors that grow rapidly there is a compression of the capillaries and a loss of function of those parts of the brain where they are located. On the contrary, in the vicinity of slowly-growing tumors there is atrophy of the brain-substance, and its shrinkage supplies as much room as is lost by the growth of the tumor. Under these circumstances the capillaries of the affected part of the brain are not compressed, and its function not disturbed. Perhaps the presence or absence of local symptoms (herdsymptome), in tumors of the cere-

brum having the same location and equal size, depends partly on whether the tumor has displaced the brain-filaments or has developed at their expense and has substituted them. My colleague, Prof. *Schüppel*, who has given me some other valuable points on the histology of cerebral tumors, asserts that the different forms of tumors vary in the above respect. Naturally, tumors, rich in vessels which increase and diminish in size according to their fulness, are not apt to run their course without symptoms; but, as a rule, are at least occasionally accompanied by symptoms of irritation and paralysis. Lastly, I shall mention that tumors running a perfectly latent course are proportionately more frequent than those having the symptoms of a severe inexplicable cerebral disease. In abscesses of the brain, just the opposite is true. This difference is probably due to the entire nutrition of the brain being generally more severely impaired by the presence of an abscess than by a tumor.

Even those tumors of the brain of which we succeed in making a certain diagnosis during the course of the disease, do not always begin with the symptoms which play the most important part in the diagnosis, that is, with the partial paralysis and anæsthesia, which we have designated as local symptoms (*Herdsymptome*), and which we have repeatedly and fully described. In many cases the first, and often for a long time the only, symptom indicative of brain-disease, is a severe headache. Although this occurs during the most different brain-diseases, and although we may even say there is no disease of the brain which is not, under some circumstances, accompanied by it; still there is no cerebral disease where continuous, unusually intense, and severe headache is so prominent a symptom, and there is no other disease where it is so often observed as in tumor of the brain. The cases where patients with cerebral tumors do not complain of headache are such rare exceptions, that its absence in a doubtful case rather speaks against a tumor. The fact that headache occurs even in those tumors of the brain which are located at a distance from the sensitive parts, supports the view we advanced, that in cerebral diseases the pain in the head started from the filaments of the trigeminus supplying the dura mater. We must be very careful about localizing the tumor from the seat of the pain. Only pains constantly confined to the back of the head permit us to conclude that very probably the tentorium is stretched, and that the tumor is probably situated in the posterior cranial fossa.

In many patients, attacks of dizziness and vomiting accompany the headache, particularly the severe exacerbations which occur, from time to time, with or without perceptible cause. This may facilitate the correct interpretation of the headache, and show its dependence on

disease of the brain, if it should have been doubtful, in spite of the duration, severity, and obstinacy of the pain.

Among the local symptoms of tumors of the brain, those of partial irritation (hyperæsthesia, neuralgia, indefinite sensations of formication, twitching, etc.) very frequently precede the partial anæsthesias and partial paralyses, and there are cases of cerebral tumors where these symptoms, and not the headache, open the scene. We have seen that partial symptoms of irritation, occurring in severe structural diseases of the brain, are only to be regarded as secondary effects, and are referable to disturbances of circulation in the vicinity of the affected part. Also that they occur in the most varied diseases, and alone are not pathognomonic of any of them. However, in cerebral tumors, there is one circumstance which is characteristic of the hyperæsthesia, neuralgia, and twitchings, and the anæsthesia and paralysis which usually follow them, namely, that these local symptoms are observed more frequently, even among the cerebral nerves, than in any of the previously-described forms of brain-disease. But we cannot say that symptoms of irritation and paralysis of the cerebro-spinal nerves, such as hemiplegia, etc., are rare, or do not occur; but only that, apart from other symptoms, the occurrence of neuralgia, hyperæsthesia, or anæsthesia, or of spasms and paralysis in the parts supplied by the cerebral nerves, goes to prove, in doubtful cases, that there is a tumor in the brain. The simple explanation of this peculiarity is as follows: In all the local diseases hitherto treated of, in necrotic softening of the brain, in cerebral hæmorrhage, in encephalitis, and its results, the morbid process is, with rare exceptions, limited to the brain, and does not attack the nerves passing from it. The case is quite different with tumors, which not only often pass from the brain to the nerves originating from it, but which, in many cases, start from the meninges or skull, and then not unfrequently destroy the cerebral nerves before attacking the brain. Let me call attention to another point, which was first published by my friend *Ziemssen*, at that time assistant in my clinic. Most paralyses of cerebral nerves are *peripheral*, when due to tumors, whether these have originated in the brain, or have advanced to the brain from the meninges or cranial bones. On the other hand, most of the few cases of paralyses of cerebral nerves occurring in the other forms of brain-disease are of *central* origin. Now, it is well known that the state of the nerves and muscles, on electrical irritation, is a certain means of distinguishing central and peripheral paralyses. In central paralysis, the muscle contracts normally when an induced current is passed through the nerve; in peripheral paralysis, on the contrary, the contraction does not take place. Hence we may thus amplify the above proposition: In cases of local disease of the brain,

paralysis in the parts supplied by the cerebral nerves (especially if the paralyzed muscles do not contract on passing an electric current through their nerve) renders it very probable that there is tumor of the brain. The assertion that, other things being equal, paralysis of a cerebral nerve favors the idea of a tumor, and that most paralyses of cerebral nerves are of peripheral origin, is not actually true of the facial nerve. Paralysis of the facial, as one symptom of hemiplegia, occurs just as often in other local diseases of the brain as in cases of tumor, and is unmistakably of central origin. What was said above is not true of these cases, but of the other peripheral facial paralyses. Next to the facial, among the motor-cerebral nerves, the oculo-motor and abducens are most frequently attacked. Paralysis of the pars minor trigemini is rare; this is also true of complete paralysis of the hypoglossal and motor filaments of the glosso-pharyngeal; while incomplete paralysis of these nerves, as shown by disturbances of articulation and deglutition, is rather common. Peripheral facial paralysis, due to tumors of the brain, is occasionally preceded by twitching of the facial muscles; while twitching of the muscles of the eye precedes paralysis of the oculo-motor, which is characterized by dilatation of the pupil, ptosis, disturbance of mobility of the eye; often, also, by diplopia and strabismus. If the oculo-motor be unaffected, paralysis of the abducens induces diplopia and strabismus convergens. Before the destruction of the filaments of the trigeminus has caused anæsthesia of the half of the face, of the conjunctiva, mouth, and nose, most patients complain of severe pain in all the parts supplied by the nerve, and not unfrequently these pains continue during the anæsthesia (anæsthesia dolorosa). Hardness of hearing, or complete deafness, from destruction of the acousticus, is usually preceded, for a time, by troublesome noises in the ears. Disturbances of vision, even to complete blindness, are very frequent in tumors of the brain, but they are not, by any means, always due to direct lesions of the optic nerves, chiasm, tractus opticus, or corpora quadrigemina. Not unfrequently, the tumor is at a distance from these parts, as in the cerebrum or cerebellum. I consider it a mistake to suppose that, in such cases, the pressure has extended, through the intervening substance, to the optic nerve, and, by pressing this against the base of the skull, caused its atrophy. The correctness of this view appears to be opposed, among other things, by the fact that, in blindness from tumors in the cerebrum or cerebellum, the motor nerves of the eye, which are under about the same conditions as the optic nerve, are rarely paralyzed. Many of the cases of amblyopia and amaurosis, caused by cerebral tumors, depend on venous congestions in the eye, and the consequent structural changes in the retina and optic nerve, and are due to com-

pression of the cavernous sinus and obstructed escape of blood from the veins of the eye. Tumors in the posterior cranial fossa only have this effect after they have caused abundant effusions in the ventricles, by compression of the openings of the venæ Galeni into the straight sinus. Since tumors, which destroy the optic nerve, very rarely spare the oculo-motor, I consider the presence or absence of disturbances of mobility of the eye as an almost certain means of distinguishing whether the amaurosis, caused by cerebral tumor, is due to destruction of the tractus opticus, chiasm, optic nerves, or to interference with the intraocular circulation. Ophthalmoscopy also gives important information on this point. The changes of the optic nerve, in blindness from cerebral tumors, as revealed by the ophthalmoscope, and the conclusions that may be deduced from them, are about as follows, according to *Graefe:*

1. Simple swelling of the optic papilla, with great tortuosity of the vena centralis. It is indicative of obstructed venous circulation, and, in its purer forms, is most frequently seen with tumors that encroach on the cavity.

2. Slight inflammatory swelling of the optic papilla, with less distinct venous hyperæmia and inflammatory participation of the retina. It occurs as a result of an inflammation of the perineurium, and of the optic nerve itself, which extends to the retina (neuritis descendens), and accompanies those intracranial processes, which, from their anatomical character or location, are suited to induce irritation in the parts above named.

3. Atrophy of the optic nerve. This may occur primarily, from direct lesion of the optic nerve by a tumor, meningitis, etc., or secondarily, as a final result of either process.

The partial disturbances of sensibility and motility in the parts supplied by the cerebro-spinal nerves, that occur in cerebral tumors, do not differ from those accompanying other local affections of the brain. Neuralgic pains, or indefinite sensations of prickling, formication, furriness, etc., as well as complete anæsthesia, occur, both over large surfaces and limited to very narrow bounds. The same is true of muscular twitchings, of contractions, and paralyses, although, among the latter, hemiplegia far exceeds the other paralyses.

If there be morbid symptoms in the parts supplied by the cerebro-spinal and cerebral nerves at the same time, they almost unexceptionally show the peculiarity of being on opposite sides of the body. This "alternation" is simply because the paralysis, anæsthesia, etc., of the cerebro-spinal nerves are caused by a lesion of the nerve-filaments before they cross, while those of the cerebral nerves depend on destruction of nerve-filaments that have already crossed.

In accordance with the slow growth of most cerebral tumors, the development and extension of the local symptoms of irritation and paralysis are usually slow and gradual. Many patients cannot state exactly when their disease began. In doubtful cases this may decidedly aid in the diagnosis, since, from such a course, we may exclude most other local diseases of the brain. But not very rarely these local symptoms come on suddenly; this is chiefly the case where vascular tumors are suddenly increased in size by overfilling of their vessels, or by hæmorrhage from them, or where the parts around them are attacked by inflammation or softening, or by capillary hæmorrhages. If the tumor have previously run a latent course, and these symptoms of paralysis come on suddenly, the case will most probably be mistaken for one of cerebral hæmorrhage, and there are numerous instances where the best diagnosticians have made mistakes in such cases. If, on the other hand, there has been a suspicion of tumor of the brain, the frequency of such incidents in the course of this disease should prevent our being deceived.

Attacks of epileptiform convulsions occur more frequently from tumors of the brain than from any other local disease of that organ; but it is very remarkable and inexplicable that they occur almost solely when the tumors are in the cerebrum, and especially when they are near the cortical substance.

In many cases psychical disturbances do not present themselves throughout the disease. Indeed, it seems that the psychical functions of the brain only suffer when the cortical substance of both hemispheres is affected by organic disease, or by disturbance of circulation. The correctness of this view is proved by the peculiarities of those cases where the state of the psychical functions forms an exception to the above. For the general mental ruin that we described when speaking of abscess of the brain also occurs in cerebral tumors, when they are accompanied by extensive chronic meningitis of the convexity, or when the cortical substance of both hemispheres is the seat of numerous tumors (cysticerci), or when their capillaries are compressed by encroaching tumors in both hemispheres, or by extensive secondary effusions in the ventricles.

In the rare cases where the tumors perforate the skull, the symptoms above described are accompanied by other symptoms, which are very characteristic. The perforation usually takes place through the parietal or temporal bones, near the root of the nose, more rarely through the occipital bone. At the point of perforation the hair is lost, the skin becomes red, and is traversed by varicose vessels; ulceration of the integument is rare. We may often feel a bony margin at the border of the tumor, which is generally soft and nodular. It is a

characteristic but not a constant symptom for the tumor to move with respiration; occasionally it may be replaced. Attempts to do this, which suddenly contract the space in the skull and prevent the entrance of arterial blood to the brain, usually induce loss of consciousness and convulsions.

The nutrition, general condition, and the functions of the body independent of the brain, often show no anomaly for a long time. In other cases it seems as if the organic processes went on with a certain sluggishness: the heart and pulse beat slowly, respirations are rare, the bowels constipated, secretion of urine scanty, and, perhaps as a consequence of this, the body often increases remarkably in size. In other cases, on the contrary, marasmus occurs early, the patients emaciate, the skin becomes dry and scaly, the mucous membranes, especially the conjunctiva, are affected with blennorrhœa. Lastly, there are bed-sores and dropsy of the feet.

The course of cerebral tumors shows many variations which we shall not discuss in detail. At first, there are generally exacerbations and remissions of the symptoms, which subsequently become constant and steadily grow worse.

In carcinomatous tumors the disease usually lasts only a few months, but occasionally a year or more. Non-carcinomatous tumors often run on for several years.

The most usual, and perhaps the only, termination is death. If it be not induced early by complications or intercurrent disease, the symptoms of general limitations of the space in the skull become more and more marked. The patients fall into deep coma, followed by death.

It is rarely possible to recognize the seat and size of a tumor exactly; but it may often be approximately decided in what part it is located, whether in the cerebrum, medulla oblongata, or in the cerebellum, at the base or at the convexity. The "brilliant diagnoses," where the precise localization of a tumor is fully confirmed by autopsy, are not usually due to acumen of the observer, but are cases of lucky diagnosis. If a basilar tumor destroys the function of several cerebral nerves, one after the other and in regular sequence, while it spares neighboring nerves, any one, having a knowledge of only the coarse anatomy of the brain, can decide the seat and extent of the tumor with almost absolute certainty. However, such cases, which are published with much self-satisfaction, are exceptions, as said before.

It would occupy too much time to speak fully of all the factors, besides the participation of the different cerebral nerves, which are to be considered when deciding the location of the tumors. We must limit ourselves to the following short hints. The presence of hemiplegia

indicates that the tumor is either in one side of the cerebrum (the most frequent seat) or in one crus cerebri, in one side of the pons or in one side of the cerebellum. In tumors of the cerebrum the hemiplegia is usually pure, that is, the other half of the body is entirely free from paralysis. In tumor of one crus cerebri, the paralysis of the opposite side of the body is almost always accompanied by paralysis of the oculo-motor of the same side. In tumors of the lateral portions of the pons, besides the hemiplegia and very common anæsthesia of the opposite half of the body, there is usually facial paralysis or anæsthesia of the side of the face corresponding to the tumor. In tumors of the cerebellum, as we have previously shown, hemiplegia is not constant, and when present is not pure, but extends to the other side of the body to a less extent, especially affecting the muscles of the spinal column. Paresis of the muscles inducing bending, erection, and lateral movements of the spinal column, first shows itself by a peculiar form of dizziness, which we have already fully described, and referred to vibrations of the spine in walking and similar acts; subsequently, it often evinces itself by an utter inability of the body to maintain itself, so in the sitting position the patients collapse and cannot move the body, unless held by both shoulders. Paralysis of both sides results from tumors in both sides of the cerebrum, in the middle parts of the pons and in the medulla oblongata. In the above description of the different symptoms it has already been shown that epileptiform convulsions indicate that the tumor is near the cortical substance of the cerebrum; pain in the back of the head shows that it is in the posterior cranial fossa; and severe psychical disturbance either indicates numerous tumors in the cortical substance of both hemispheres, or that there is secondary meningitis or hydrocephalus. It appears to me that too little attention has been paid to the fact that the intelligence which is at first intact is very much impaired in the later stages of tumors encroaching on the posterior cranial fossa, which impede the escape of blood from the ventricles and induce extensive transudations into them.

Even on most carefully weighing all the symptoms, many errors occur, and it is very desirable that observers, who have access to a large number of cases, should publish their errors of diagnosis, as well as their successes, more than has hitherto been done. The trustworthiness of the different aids to the diagnosis and localization of cerebral tumors cannot become perfectly clear till this is done.

The wide-spread error of designating the various forms of neoplasia, aneurisms of the brain, and even parasites, by the common name of cerebral tumors, is undoubtedly due to the fact that there are very few cases where the nature of the tumor can be certainly determined

during life. If the symptoms of cerebral tumor occur in a person who shows signs of carcinoma, tubercle, aneurism, or parasites in some other organ, we are justified in presuming that the cerebral growth is of the same nature as the other tumor, neoplasia, or parasite; but this aid to diagnosis fails in most cases, except where there are tubercles of the brain in persons with tuberculosis of the lungs, and syphilomata of the brain in persons with the same affections of other organs. Carcinoma of the brain is, as a rule, primary and solitary; aneurism of a cerebral artery is usually the only one in the body, and, when cysticerci and echinococci enter the brain, as a rule they also are limited to that organ.

The age of the patient gives some indication as to the nature of the tumor. Most frequently children are affected with tubercle, young adults with benignant tumors, especially sarcoma and glioma, and persons of advanced years with carcinoma. In his classical observations on brain-diseases, *Griesinger* has shown that, under some circumstances, it is possible to make a diagnosis of cysticerci in the brain, even when there are none in other parts of the body. The large number of these small parasites, and their customary seat in the superficial layers of the cerebrum, explain the severe psychical disturbances and the epileptiform attacks, while the absence of a large compressing body explains the non-occurrence of symptoms of paralysis, on which factors *Griesinger* founded the diagnosis in his case. On the other hand, I regard *Griesinger's* experiment, of compressing both carotids firmly against the transverse processes of the cervical vertebræ, as entirely useless for the diagnosis of basilar aneurism, and he, too, says it is "purely theoretical." The occurrence of convulsions during this act is not at all significant of an aneurism, even of an obliterated one. In many other diseases, also, where there is any thing encroaching on the intracranial space, compression of both carotids causes dizziness and loss of consciousness with or without convulsions.

Treatment.—We can do but little to remove carcinoma, sarcoma, and the neoplasia generally, in other organs, by the hunger-treatment, by the administration of iodine, arsenic, etc., and these remedies are of no more benefit in tumors of the brain. They hasten the fatal result instead of retarding it. The treatment can only be palliative and symptomatic. It is useless, and annoys the patient, to apply a seton or issue to the nape of the neck. On the contrary, it is advisable to guard him in every way from hyperæmia of the brain, by which acute swelling of the tumor and threatening attacks are most frequently caused; to regulate the nutrition and mode of life; to attend to the bowels, etc. Apoplectic or inflammatory attacks must be combated by venesection, local bleeding, ice-compresses, etc., as before

advised. When the pain in the head is unusually severe, we may order local abstraction of blood and cold-compresses or derivatives to the nape of the neck. If these fail, we should not be too timid about giving narcotics, and particularly small doses of morphia. When there is the slightest suspicion of syphiloma of the brain, instead of this symptomatic treatment, we should institute an energetic antisyphilitic course. It is unpractical to delay this treatment, or to neglect it altogether, if the syphilitic nature of the disease be not accurately determined. Experience teaches that even very severe structural changes are capable of recovery, and often disappear under proper treatment, and, on the other hand, that treatment is powerless against carcinoma, sarcoma, etc. Hence, we risk little and may gain much, if, on the mere suspicion of syphiloma of the brain, we treat the patient as if there was no doubt about the diagnosis.

CHAPTER XIV.

SEROUS EFFUSIONS IN THE MATURE SKULL—HYDROCEPHALUS ACQUISITUS.

We have already spoken of one form of hydrocephalus, that which almost constantly complicates basilar meningitis, and, in the next chapter, when treating of congenital hydrocephalus, we shall also treat of the effusions occurring shortly after birth, before the sutures are closed. In the present chapter we shall only consider those serous effusions into the cranial cavity which occur without basilar meningitis and after the skull has closed.

Etiology.—Serous effusions into the arachnoid sac (hydrocephalus externus) are rare, and hardly ever become extensive. Effusions into the subarachnoid space and ventricles and œdema of the brain are more frequent.

Hydrocephalic effusions are due partly to increased lateral pressure in the vessels, partly to an abnormally slight amount of albumen in the blood, partly to disturbances of nutrition, which render the walls of the vessel more permeable. As one symptom of general dropsy, whether dependent on disturbance of circulation or on abnormal quality of the blood (morbus Brightii), the hydrocephalus rarely attains a high grade, although, perhaps, some sudden deaths occurring in the diseases inducing the dropsy are to be referred to slight effusions in the ventricle and to a slight œdema of the brain. Tumors and other diseases in the posterior cranial fossa, by compressing the venæ Galeni or straight sinus, and obstructing the escape of blood from the ventricle, sometimes induce high grades of hydrocephalus. Hydrocephalus, de-

pendent on certain disturbances of nutrition in the capillaries, is more of an independent disease than the other forms; it closely resembles the inflammatory processes, and may be ranked with the inflammations of the skin that cause serous blebs. This view is supported by an interesting observation of *Hoppe*, who, on examining the fluid from a chronic hydrocephalus, found it differently constituted from the normal cerebro-spinal fluid, particularly that it contained more albumen. This form of hydrocephalus occurs chiefly among children, and, when seen later in life, it almost always dates from childhood.

We must mention, as a peculiar variety of the disease, *hydrocephalus ex vacuo*, which develops as a necessary result of diminution of the size of the brain, whether from general, especially senile, involution (hydrocephalus senilis), or from partial atrophy of the brain.

Anatomical Appearances.—The amount of fluid effused into the arachnoid sac is difficult to decide, since, on removing the brain from the skull, the fluid from the subarachnoid space is always mixed with it; but, as we have before said, it never becomes considerable.

The fluid effused in the subarachnoid space is sometimes regularly, sometimes irregularly spread over the surface of the brain; in the latter case the arachnoid often forms a vibrating sac filled with serum. When slight, œdema of the brain shows itself by the moist lustre of the cut surface; when more decided, the coherence of the brain is decreased, and, if we press on some part, the resulting pit is filled with liquid after a time. When of very high grade, the brain-substance is broken down by the œdema, and changed to a thin white pulp (hydrocephalic softening).

In acute hydrocephalus internus the fluid effused into the ventricle rarely exceeds half an ounce or an ounce; it is either clear, or, as is usually the case, slightly clouded by a scanty admixture of cast-off epithelium, flocculent clots, and fragments of the surrounding brain-substance. The walls of the ventricle, especially the septum, fornix, and commissures, are in a state of hydrocephalic softening.

In chronic hydrocephalus internus we generally find the ventricles considerably dilated: they may contain ten or twelve ounces of fluid, which is usually clear. The ependyma of the cerebral cavities is thickened and often strewn with fine granulations. The surrounding brain-substance is also more dense and tough.

Symptoms and Course.—Of course, the cavity of the skull is as much encroached on by serous effusions as by extravasations of blood or products of inflammation. As a necessary result of this, we have mentioned obstruction to the entrance of arterial blood, and have given attacks of convulsions and loss of consciousness as the symptoms of complete and sudden arterial anæmia; general symptoms of irritation

and depression as those of incomplete and gradually-developing anæmia. If, at the same time, we remember that those portions of the brain bounding the ventricles are destroyed, in acute hydrocephalus, by softening, in the chronic form by atrophy, and that this may induce partial paralysis, we may construct a picture of the disease, either in the acute or chronic form, exactly corresponding with direct observation.

Sudden and large effusions of serum into the cerebral substance and ventricles lead to a combination of symptoms which, from its resemblance to the apoplectic attack induced by extravasation of blood, is usually termed apoplexia serosa.

After what was said above, it is hardly worth mentioning that a distinction between serous and sanguineous apoplexy is not by any means possible in all cases, and that, if a diagnosis be made, it is chiefly from the etiology, which indicates one form rather than another.

In children, acute hydrocephalus almost always runs its course with the symptoms that we ascribed to high grades of cerebral hyperæmia, and to the first stages of acute meningitis. Severe attacks of convulsions, with loss of consciousness, are the most frequent and characteristic symptoms. If these attacks occur very often, and last unusually long, they should excite the fear that the hyperæmia has induced considerable transudations in the ventricles, and it is to be feared that the transudation will not be absorbed, or, at least, only partly so, and that chronic hydrocephalus will remain.

The symptoms of chronic hydrocephalus either succeed those of the acute form, or they develop insidiously and gradually. They consist of headache, dizziness, weakness of the special senses, particularly of the eyes, very often of a general paresis, preceded by a tottering gait and trembling of the limbs. Among the constant symptoms are disturbances of the intellect, especially its gradual loss, even to idiocy. This is sometimes accompanied by convulsions, and occasional vomiting. The patients usually have a slow pulse, are readily chilled, are occasionally ravenous; they have a puffy look, and varicose vessels on the cheeks. Of course, these symptoms only render the diagnosis of chronic hydrocephalus certain when other cerebral diseases, accompanied by similar symptoms, can be excluded, and, consequently, that the diagnosis can rarely be made with absolute certainty. The course of the disease is usually very tedious. If death do not sooner occur from some intercurrent disease, it finally results, sometimes rapidly and unexpectedly, from acute increase of the effusion, sometimes slowly, from its gradual increase, being usually preceded, for some days, by continued deep sopor.

Treatment.—The treatment of acute hydrocephalus is the same as that of acute hyperæmia of the brain and acute meningitis. In

chronic hydrocephalus, the continued employment of cold douche baths deserves most confidence.

CHAPTER XV.

SEROUS EFFUSIONS IN THE INCOMPLETE SKULL—HYDROCEPHALUS CONGENITUS.

Etiology.—It is most probable that congenital dropsy of the brain is due to inflammation of the ventricular walls that has occurred during fœtal life. The etiology of the disease is entirely obscure. Some women have had several hydrocephalic children, without any apparent cause.

Anatomical Appearances.—In congenital hydrocephalus the amount of serum, usually clear and limpid, effused into the cerebral cavities, is occasionally very small, while it sometimes amounts to six or ten pounds. In such cases the ventricles are distended to large, thick-walled sacs, the brain-substance around them is thinned, and often atrophied to a layer only a few lines thick; the corpora striata and optic thalami are flattened and pressed apart, the corpora quadrigemina flattened, the commissures stretched and thinned. The septum is occasionally broken through, and the floor of the third ventricle is often thinned and projecting. The pons and cerebellum appear compressed from above downward.

Where the effusion is not large, the skull preserves its normal size; where it is large, on the other hand, it is almost always decidedly distended. Even at birth the head is usually enlarged, but becomes still larger after birth, and may be distended to two feet in circumference. In such cases the cranial bones, especially the frontal and parietal bones, are very large, and are, at the same time, very thin. The membranous interstices also, especially the fontanels, are very wide. The forehead is prominent, the roofs of the orbits are depressed, and they are transformed into narrow, transverse slits; the squamous portions of the temporal bones and the occipital bone are more horizontal. The ossification, which is delayed, takes place from formation of points of ossification in the membranous interspaces, or even from the formation of innumerable small bones. The cranium, which, previous to the ossification, was very thin, often becomes thick subsequently, and, in many cases, such a skull retains an asymmetrical or remarkable spherical form.

Symptoms and Course.—Many children, born with hydrocephalus, die at birth or shortly after. In others, during the first weeks of life, if the skull be not enlarged, no symptoms of the disease can be observed. This is due partly to the difficulty of judging of the cere-

bral functions at this age, partly to the yielding nature and gradual distention of the skull. If an equal amount of transudation occurred in the mature skull, the severest symptoms would arise.

Even during the first years of life, the increasing size of the skull and the growing difficulty that the child finds in holding its head upright are the most prominent and only characteristic symptoms. If the head be not enlarged, or so slightly so as not to be noticed, the disease is generally overlooked in the first year also. It is true the mother wonders that, when the child is nine months old or over, it still remains uncleanly, makes no attempts to walk, and does not even try to speak; she hardly asks the physician's advice, and, when she does finally consult him, he also reassures her. But gradually the child appears more strange. First of all, there is an idiotic manner with outbursts of pleasure or fear, in which the child often shrieks out, distorts the face into horrible grimaces, and drums with the extremities. The first year passes, and the child continues to stick all toys, for which other children of the same age seem to understand the use, into its mouth, because it does not know what else to do with them. The eye does not regard any object held in front of it, but rolls about unsteadily. The face has no expression, but is empty and silly; often saliva flows constantly from the half-open mouth. And gradually comes the sorrowful conclusion that the child does not develop intellectually, or even loses ground. Many such children do not learn to walk. If we attempt to teach them to walk, they often cross the legs instead of setting them forward. Others, who do learn to walk, have such an uncertain and helpless gait that they readily fall, and frequently cannot step over any elevation. There are usually no anomalies in the organs of special sense. On more careful observation, apparent deafness generally turns out to be deficient attention. It is difficult to come to any decision regarding the senses of smell and taste; the sense of sight usually remains intact, though there is often strabismus or dilatation of the pupil.

In those cases where the head quickly becomes very large, the disease is recognized sooner and more easily, although in them the above symptoms are usually less developed than where the head is moderately or not at all enlarged. The little old face, which does not agree with the large skull, and forms with it a triangle pointing toward the chin, the distended veins traversing the skin, especially in the frontal and parietal regions, the thin hair covering the broad skull, the often rachitic bones or a general dwarfy appearance, the vain attempts of the child to hold up the heavy head, which sinks again every time it is raised, give the disease an appearance as melancholy as it is characteristic.

Apart from the intercurrent attacks of convulsions, which are quite frequent, the course of the disease is sometimes regular and steady, so that the symptoms increase gradually till death results from general paralysis, or it is irregular, so that the symptoms are worse at times, while at other times there is a temporary stand-still or even improvement. Lastly, it is not rare for the disease, after attaining a certain point, to remain permanent, or even for part of the existing disturbances to disappear. This disappearance probably never goes on to recovery; some intellectual weakness always remains, though it may be only a slight amount.

The most frequent termination of the disease is death; this often occurs during the first years of life, partly from the disease itself with convulsions and consequent coma, partly from complications.

The increasing encroachment on the cranial cavity shows its effects earlier in the mature skull than in the opposite case, and the life of the child is in greater danger when the skull remains of the normal size than when it is considerably dilated. In rare cases death results from rupture of the distended ventricle and soft parts covering it, either spontaneously or from a fall or blow. Few patients live beyond puberty, and it is very rare for them to attain mature age.

Treatment.—In congenital hydrocephalus nothing can be accomplished by absorbent remedies, diuretics, drastics, preparations of iodine, calomel, or mercurial salve. Compression of the distended skull by strips of adhesive plaster is not free from danger, and in cases hitherto observed has proved of little use. Nor are the results of evacuating the serum by operation very encouraging. Most patients died soon after the first puncture or after its repetition, so that the operation should be limited to those cases where the head has attained a colossal size, and where a steady progress of the enlargement is observed. In the treatment of congenital hydrocephalus as in the acquired form, the treatment is mostly symptomatic, and the patients are to be protected as much as possible from all injurious influences.

CHAPTER XVI.

HYPERTROPHY OF THE BRAIN.

Etiology.—Since, in so-called hypertrophy of the brain, there is not an increase of the true tissue-elements of the brain, the nerve-filaments, and ganglion-cells, but only a proliferation of the delicate interstitial substance which unites the nerve-elements, the name hypertrophy of the brain is not exactly correct. It is sufficient, however, to have noticed this point, and we may preserve the old name

without fear of being misunderstood. We do not know whether the proliferation of the neuroglia is the result of frequently-recurring hyperæmia, or of some other cause.

Hypertrophy of the brain is occasionally congenital, and then often accompanies dwarfishness; more frequently it develops after birth, is chiefly limited to childhood, and becomes rarer with advancing years. Hypertrophy of the brain acquired after birth is usually accompanied by rachitis, deficient involution of the thymus gland, and hypertrophy of the lymphatics (*Rokitansky*). Mental excitement, abuse of liquor, and lead-poisoning, are mentioned as exciting causes of the disease, but it is very doubtful whether they act in this way.

Anatomical Appearances.—The brain, almost exclusively the cerebrum, is larger and heavier than normal. On autopsy, if the top of the skull be removed it is difficult to replace it, because the brain bulges out so, and protrudes between the sawed edges of the bone. The cerebral membranes are very thin and bloodless. We often find no trace of liquid in the subarachnoid space. The convolutions on the surface of the cerebrum are flattened and pressed together; the sulci between them are scarcely perceptible. The centrum semiovale is unusually large, the ventricles are small on section; the brain-substance, like the membranes, appears bloodless and dry. Its consistence and elasticity are increased.

If hypertrophy of the brain develop before closure of the skull, the latter is distended, just as in congenital hydrocephalus. If, on the other hand, it does not begin till the sutures are closed, the cranial wall is often thinned by absorption, and the inner lamella loses its smoothness. More rarely, when the disease increases rapidly, the closed sutures are opened and pressed apart.

Symptoms and Course.—Of course, hypertrophy of the brain has the same influence on the intracranial circulation as any other increase of the contents of the skull has; hence it is accompanied by extensive symptoms of irritation and of paralysis. And it may readily be understood that these symptoms often do not occur, or only attain a low grade, as long as the sutures of the skull are not closed, and the skull can distend in proportion to the enlargement of the brain. The circumstance that children with hypertrophy of the brain are not always deficient in intellectual development, but are sometimes remarkably sharp and competent in proportion to their physical development, forms a strong point in diagnosis between hydrocephalus and hypertrophy of the brain in children with very large heads. Among the symptoms most frequently seen, when there is no enlargement of the head, or when it does not correspond to the increase of the brain, the most important are attacks of epileptiform convulsions. These at-

tacks seem to occur principally when the one constantly present cause, arterial anæmia, is accompanied by any other, often a very slight one, which suddenly and temporarily greatly increases the anæmia. Headache, dizziness, photophobia, general hyperæsthesia, mental irritability, occasional vomiting, and subsequently anæsthesia, general muscular debility, mental hebetude and drowsiness, are much less frequent and far less characteristic of the disease. The malady can only be diagnosed with an approach to certainty where the skull is enlarged and hydrocephalus can be excluded, while in most cases only a probable diagnosis can be made. The course of hypertrophy of the brain is always chronic. It is doubtful whether it ever ends in recovery. Death results either from the disease itself, generally not from a gradually increasing paralysis, but during a severe attack of convulsions, or else from complication with hæmorrhages and inflammatory exudations; as may readily be understood, even when very slight, these are extremely dangerous for such patients.

We cannot speak of treatment of hypertrophy of the brain, for, even where the disease is recognized, we cannot expect any remedy to remove the existing disturbance.

CHAPTER XVII.

ATROPHY OF THE BRAIN.

Etiology.—We must not term every diminution of the brain-substance, particularly those due to destruction of its tissue and to development of shrinking cicatricial tissue in the place of the destroyed portion, atrophy of the brain. True atrophy of the brain consists rather in a diminution of the size or number of the elements, without any perceptible previous destruction of them.

It is convenient to distinguish two forms of atrophy of the brain. In the first, the so-called agenesis, there is incomplete development; in the second there is a retrogression, a disappearance of the previously well-developed constituents of the brain.

We pass over those forms of agenesis where the brain is so incompletely developed that there is either complete idiocy or that life cannot continue, and turn our attention to the interesting and not very rare form of one-sided atrophy of the brain, occurring during fœtal life and the first years of childhood, in which not only may life continue, but there may be a certain amount of intellectual development. The etiology of monolateral agenesis is obscure. It is probably due to inflammation of the brain, meninges, or skull, during fœtal life or early childhood.

The atrophy occurring after complete development of the brain is sometimes primary, sometimes it accompanies other affections of the brain as a secondary disease. Among the primary atrophies we must first mention that form which occurs as a symptom of senile marasmus. Other senile changes attain unequal grades in different persons, and we also meet very aged individuals who do not show the least sign of cerebral atrophy, while it advances to the highest grade in much younger persons. Next to this comes the atrophy of the brain, which develops in the course of exhausting and consuming diseases. In many cases which are quoted as examples of great resignation and wonderful firmness at the approach of death in tedious diseases, the facts were really badly interpreted; very often the resignation is certainly due to the dulness and apathy induced by cerebral atrophy. Local diseases of the brain are the chief causes of secondary atrophy. We have already mentioned it as the result of precedent apoplexy, partial necrosis, and partial encephalitis. The paralytic form of idiocy appears to depend on an atrophy of the brain caused by chronic meningitis or inflammatory processes in the cortical substance. In other cases the atrophy is the result of continued pressure on the brain. Under this head come the cases where the size of the brain gradually decreases under the pressure of cerebral tumors and hydrocephalic effusions. In meningitis, also, perhaps part of the atrophy is due to the pressure of the inflammatory exudation in the subarachnoid space. Lastly, we must mention that injury and destruction of peripheral nerves occasionally induce secondary atrophy of their centres.

Anatomical Appearances.—When cerebral agenesis is limited to one side, the left is the one generally affected; sometimes the whole hemisphere is affected, sometimes only parts of it. In high grades of the disease, the cerebral substance between the ventricles and convex surface has become a thin layer only a few lines thick. The convolutions are scarcely perceptible, or else are very small. The large cerebral ganglia are usually atrophied, and the atrophy extends from them through the crura cerebri to the spinal marrow. The consistence of the atrophic brain is usually increased, its color is somewhat dirty. The space created by the atrophy is filled with fluid that has collected partly in the ventricles, partly between the meninges. The skull is often unsymmetrical, and is thickened at the atrophied part.

Atrophy of the brain occurring late in life is usually total, but, when it results from partial destruction of the brain, it is generally further advanced on the side corresponding to the disease than on the other. The medulla of the cerebrum is diminished, the convolutions appear thinner, the furrows broader and deeper. The medullary substance is dirty white, more dense and tough, the cortical substance is

thinner, harder, and of a pale or light-brown color. The ventricles are dilated and filled with serum. There is also a quantity of fluid in the subarachnoid meshes (hydrocephalus ex vacuo).

Symptoms and Course.—Although, as we have previously mentioned, agenesis of one side is not always accompanied by psychical disturbances, still it is rare to find cases where we dare assert that one side of the brain fully replaces the other. Far the greater number of patients suffer from weakness of intellect, and many from decided idiocy. The organs of special sense, particularly the eye, are usually very insensitive, and the excitability of the sensory nerves of the paralyzed half of the body is diminished. The most important and apparent symptoms are paralysis and a peculiar and excessive atrophy of the side of the body opposite to the atrophied brain. The paralysis is not usually complete, so that the patients can generally perform some imperfect movements. The paralysis is ordinarily combined with contractions. The atrophy of the paralyzed side affects all the tissues, the bones not excepted, so that the thin and short extremities of a grown person appear like those of a child. Most patients suffer from epileptic attacks. As the other functions of the body are usually well performed, the disease itself is rarely fatal. But the patients seldom attain old age. Their power of resistance to intercurrent diseases is lessened, and they succumb to them more readily than other persons would.

The primary atrophy of the brain, which develops chiefly in old persons, and secondary atrophy, which accompanies apoplexies, partial necrosis, and other local brain-diseases, are characterized by gradual weakening of the psychical functions, loss of memory, slowness of thought, absent and childish manners, as well as dulness of the senses, and gradual weakening of the motor power, unsteadiness of motion, trembling, incomplete control of the sphincters, etc.

The atrophy of the brain found on autopsy of insane patients, who have suffered from paralytic idiocy, belongs to the terminal symptoms observed in that form of insanity during life, to the symptoms of mental weakness and idiocy, but not to the monomania which preceded the mental paralysis, or to the intercurrent maniacal and apoplectiform attacks. These are rather to be referred to the precedent meningitis, which is rekindled from time to time during the subsequent course. At the stage of the disease when the psychical exaltation of the patient decreases, when the insane ideas and hallucinations lose their richness and reality, when the thoughts are confused, memory defective, symptoms of paralysis also begin to appear in the motor sphere; and the more the signs of psychical weakness increase, the more extensive and marked become the signs of motor paralysis. The

first symptom of the latter is always impaired articulation, which gradually becomes sheer mumbling. The attitude is negligent, the gait uncertain and tottering, the patient easily falls, and his hands tremble when held out. Later, he cannot leave the bed, lies motionless, does not react to the strongest irritation, and finally dies of marasmus.

Treatment.—We cannot expect to treat atrophy of the brain successfully. Treatment must be directed against the original disease, to prevent the progress of the atrophy, if possible. On this occasion we shall again call attention to the cold douches which we recommended as peculiarly efficacious in chronic meningitis. For the rest, we have to limit ourselves to combating the more threatening symptoms. In monolateral agenesis, we may attempt to arrest the atrophy and fatty degeneration of the muscles by using the induced current of electricity, which, in this case, is of course to be regarded only as a gymnastic remedy.

[CHAPTER XVIII.

APHASIA—ASYMBOLIA.

There is a disease of the brain which, while it impairs or even totally destroys the power of speech, does not in the least disable the apparatus for speaking, nor derange the faculty of thinking. This incapacity to express thoughts in words, although the mind be clear and the muscles of speech in good order, is called *aphasia*. *Trousseau* and *Broca* observed that such disorders of speech stood in close connection with a lesion of the third frontal convolution, and that nearly always an affection of the left third, and quite exceptionally a similar one of the corresponding convolution of the right side, was at the root of failure of speech. This relation the French authors first explained by assuming that in most persons the organ of speech is developed only upon one side, and that usually the left one ; while among left-handed persons the right third convolution should represent the function of speech. Besides the third convolution, however, there seem to be other spots in the brain which have to do with the speaking faculty ; and in a good many cases the diseased point has been found elsewhere, in the fissure of Sylvius, the island, or nearer to the corpora striata, or even in the posterior lobes of the brain.

The lesions which may cause aphasia are various—apoplexies,

inflammatory and embolic points of softening, injuries of the head followed by inflammatory growths, and the like. When the lesion is extensive, of course other functional derangements of the brain will accompany the aphasia. These coarse structural changes, however, are not the only causes of aphasia. It has appeared during the course of acute infectious disease, such as typhus, and as a consequence of poisoning by lead, belladonna, and chloroform. It has also arisen in hysteria and epilepsy, as well as after violent mental emotion.

Generally only one or two words remain at the command of an aphasic person. With these he in vain strives to express his thoughts, repeating them malapropos on all occasions. In some cases the speech is quite lost, or the patient can only utter unintelligible sounds. Although this lack of power to clothe the thoughts in words is the commonest and most marked phenomenon, yet it is not the only one. Very often the patient cannot write (*agraphia*) nor read (*alexia*); sometimes even the power to communicate ideas by gesture is also lost. Hence the term *asymbolia* (*Finkelburg*) would seem more suitable to the disease than aphasia. *Finkelburg* defines asymbolia as an affection in which the faculty of conveying ideas by means of signs, or else the ability to understand such signs, is impaired or lost. Indeed, aphasic persons are very often incapable of reading or understanding either written or printed words. In very exceptional cases the speaking faculty has been preserved, while the ability to make or to comprehend signs has failed. To this condition the term aphasia certainly does not apply. An aphasic patient now at the Giessen clinic for some weeks presented the odd peculiarity of being unable to open his mouth when told to do so. He would make awkward and vain attempts to open it with his fingers; but the moment a spoonful of food was presented to his lips, the mouth would gape to the verge of its ability.

We have not as yet any satisfactory explanation of the physiology of aphasia. *Trousseau* ascribed the loss of speech to two conditions: 1. To partial lack of memory. In such cases the patients do not speak because they cannot remember the words; but they can still repeat words spoken to them (*amnestic* form). 2. To forgetting the art of articulation, so that repetition of words spoken to the patient is difficult or impossible to him (*atractic* form).

It is very easy to recognize an uncomplicated, well-pronounced case of aphasia; but the diagnosis of an ill-marked case, or of one combined with other mental disorders, may be very difficult. Hemiplegia often exists on the side opposite to that of the brain-lesion (hence generally upon the right side); and since the aphasia in such

cases probably depends upon destructive local disease of the brain, the prognosis should be unfavorable, although improvement may perhaps take place. When there is no paralysis, and when we have reason to ascribe the attack to a transitory derangement of the circulation, to a watery exudation, or the like, we may hope that the affection may pass away.

Treatment of aphasia should be mainly a treatment of its cause. Later in the disease the attempt may be made to develop the speaking faculty from the opposite half of the brain, by judicious practice of reading, writing, and elocution. Such vicarious compensation is, however, not to be relied on.]

ADDITION TO THE REVISED EDITION OF 1880.

SECTION I.—DISEASES OF THE BRAIN.

1.—P. 216.

The discovery that the bursting of a vessel is preceded by an aneurismal dilatation of its walls is most important in the etiology of apoplexy. It used to be assumed that the fatty degeneration attacked only the inner and middle coats, causing an escape of blood into the adventitia, which then bulged out under the blood-pressure (*Kölliker's* and *Pestalozzi's* spurious aneurism ; *Virchow's* dissecting ectasy). *Charcot* and *Bouchard* take quite a different view of the vascular dilatation which plainly lies at the root of most apoplexies. According to them, a *periarteritis*, or inflammation of the lymphatic sheath, forms the starting-point of the process, and the morbid changes progress from without the vessel inward (the reverse of what occurs in atheroma), the adventitia thickens more or less, or at least shows a multiplication of its nuclei, while the muscular coat tends to atrophy. At points where the thickening of the adventitia has failed while the muscular coat has atrophied, the vascular wall yields before the blood-pressure ; thus there form small round knots (*miliary aneurisms*), situated on the vessels, and having the diameter of $\frac{1}{5}$—1 millimetre. If these little aneurisms contain liquid blood, their color will be red ; if the blood be coagulated, they present a brown or blackish appearance. If the thickening of the wall be considerable, the sac may attain the consistence of a grain of sand.

Zenker, while supporting the Frenchmen regarding the fre-

quence and importance of miliary aneurism, believes that they originate in a sclerosis of the tunica intima, like that which attacks the larger vessels.

Apoplexies which arise during scurvy, morbus maculosus, and malignant infectious disorders, appear to depend upon a morbid tenderness of the vascular walls caused by fatty degeneration.

SECTION II.

DISEASES OF THE SPINAL MARROW AND ITS MEMBRANES.

CHAPTER I.

HYPÆREMIA OF THE SPINAL MARROW AND ITS MEMBRANES.

There is hardly any doubt that the amount of blood contained in the spinal marrow is subject to variation, and that hyperæmia and anæmia modify its functions just as they do those of the brain. Nevertheless, the symptoms which we are in the habit of referring to an abnormal amount of blood in the spine are not directly deduced from comparison of the symptoms observed during life with the results of autopsy, but from *a priori* reasoning. Moreover, in most autopsies no attention is paid to the amount of blood in the spinal marrow, and its estimation offers even more difficulty than is the case in the brain.

Remarkable vascularity of the spinal medulla and its membranes is most frequently found on autopsy of new-born children and of persons who have died of spasmodic affections or of acute febrile diseases. We also find varicose dilatations of the venous plexus in the lower part of the spinal canal, as one of the symptoms of abdominal plethora accompanying cirrhosis of the liver and other diseases that impair the circulation in the abdomen.

Hyperæmia of the spinal medulla itself leads to swelling and relaxation of its substance, and to the formation of small ecchymoses; in higher grades there is softening of the medullary substance. According to *Hasse*, hyperæmia of the membranes induces increased transudation, as a result of which there may be overfilling of the subarachnoid space, extending from below upward as far as the skull.

As symptoms of hyperæmia of the spinal medulla and its membranes, the above author mentions a dull pain, mostly limited to the sacral and lumbar regions, a feeling of numbness and formication in the

lower extremities and their incomplete paralysis. These disturbances of sensation and motility, proceeding from below upward, rarely extend to the upper extremities; when this does occur, the respiration is also said to be occasionally affected, while the bladder and rectum hardly ever participate in the paralysis. From this combination of symptoms we cannot decide on hyperæmia of the spinal marrow with any certainty, unless there are other symptoms of it, and the symptoms disappear after bleeding from the hæmorrhoidal or uterine veins, which anastomose with those of the spinal medulla, or after local abstraction of blood. It is at least doubtful whether convulsions result from excessive hyperæmia of the spinal marrow, since the hyperæmia found on autopsy after severe convulsions may just as well be the result as the cause of the spasm. The same is true of the association between the spinal hyperæmia, found after febrile diseases, and the symptoms of the fever. "Spinal irritation," which for a time caused a good deal of talk, is a term used to indicate a condition chiefly characterized by sensitiveness of certain spinal processes to pressure, great inclination to reflex movement, and a general hyperæsthesia. We find these symptoms just as frequently as we do headache in many acute and chronic diseases, without its being possible to interpret them. At all events, we are not justified in deciding from their presence, without further proof, that there is hyperæmia of the spinal marrow, any more than we are in diagnosing cerebral hyperæmia in patients who have only headache.

If the causes of the spinal hyperæmia be made out, we should, in the first place, attempt to fulfil the causal indications. When the hyperæmia has attained a certain grade and we cannot hope for its subsidence without therapeutic aid, we may employ local blood-letting by wet cups and leeches. We should apply the former along the spinal column, the latter about the anus, especially when there is coincident abdominal plethora. The effect of the abstraction of blood is supported by cathartic medicines, particularly the neutral salts.

CHAPTER II.

HÆMORRHAGE OF THE SPINAL MARROW AND ITS MEMBRANES—SPINAL APOPLEXY.

Except small ecchymoses, which accompany excessive spinal hyperæmia, extravasations between the meninges and into the substance of the spinal marrow are very rare. Degeneration of the walls of the vessels, and increased pressure of blood in the arteries, which are the chief causes of cerebral hæmorrhages, appear to have no effect on

spinal hæmorrhage. Intermeningeal hæmorrhages may almost always be traced to injuries of the spinal meninges from wounds, contusions, or stretching. Their occurrence chiefly among the newly-born is due to the severe tension to which the spinal column of the child is so often subjected during severe labor. Extravasations of blood in the medulla spinalis are usually terminal symptoms of chronic destructive processes of the cord, and are only rarely due to injuries of the spinal column.

The extravasations of blood from meningeal hæmorrhage are usually considerable; they collect chiefly in the lower part of the spinal canal, but often fill large portions of the subarachnoid space. Where the hæmorrhage is into the medulla, we find it containing a bloody pulp. The changes undergone by the apoplectic clot, when it has existed a long time, are little known, but seem to resemble those undergone by the brain under similar circumstances.

Apparently intermeningeal hæmorrhages only gradually compress the vessels of the spinal cord sufficiently to entirely cut off the supply of arterial blood, and so remove the excitability of the nerve-filaments. In effusions of blood between the meninges, symptoms of severe irritation, pains in the back, and spasms, especially tonic spasms in the parts supplied by the nerves going off below, opisthotonos, rigid contraction of the extremities, etc., usually precede the paralysis. But where there are large extravasations, there is perfect anæsthesia, and paralysis of the parts receiving nerves from the compressed portion of the spinal cord. If the respiratory muscles be among these parts, death soon occurs; if they remain unaffected, death may be delayed. It is doubtful whether the conduction can be restored and recovery take place after disintegration and reabsorption of the extravasation. From the above symptoms we can only make a diagnosis of meningeal spinal apoplexy when they have been preceded by an injury of the spinal canal. If the history be imperfect, and we find no causes rendering hæmorrhage probable, the disease cannot be recognized with certainty.

Since the substance of the spinal marrow is usually entirely broken down by hæmorrhages into it, the conduction from the brain to the peripheral nerves, and from them to the brain, is generally interrupted at the moment of the hæmorrhage. The more sudden the symptoms of this interruption—anæsthesia, and loss of voluntary motion in the lower half of the body, combined with paralysis of the bladder and rectum—appear, the more probable it is that the communication has been interrupted by a rapid breaking down of the spinal medulla from an extravasation of blood, and not by its gradual destruction from inflammation, softening, or paralysis. When the apoplexy is high up, so

that respiration is affected, death occurs quickly. If, on the other hand, a part far down be destroyed, death does not result for years, and is generally caused by large bed-sores, or cystitis, induced by paralysis of the bladder.

Since we cannot, by therapeutic means, hasten the reabsorption of the effused blood, or aid the regeneration of the broken-down nerve-filaments, the treatment of spinal apoplexy can only be symptomatic. At first, as long as there are severe pains in the back and symptoms of inflammation, the proper treatment is local bleeding, and the application of an ice-bladder to the part where the hæmorrhage is suspected. Subsequently there is usually little to do but to guard the patient against bed-sores, to carefully empty the bladder at regular intervals, and to maintain the strength of the patient. Well-to-do persons, who remain paralyzed from spinal apoplexy, may be sent to Wildbad, Pfäffers, or Gastein. But, the more certain the diagnosis, the more improbable it is that these baths will prove beneficial.

CHAPTER III.

INFLAMMATION OF THE MEMBRANES OF THE SPINAL MARROW—MENINGITIS SPINALIS.

Etiology.—Inflammation of the dura mater probably never occurs as an independent disease, but it very frequently follows injuries, and especially inflammations of the spinal column. Acute inflammation of the arachnoid is also almost solely observed as an accompaniment of inflammation of the dura and pia mater; but chronic inflammation of the arachnoid, resulting in partial thickening and ossification, occurs as a primary and independent disease, without perceptible cause. Acute inflammation of the pia mater from epidemic influence has already been described, among the diseases of the brain, as meningitis cerebro-spinalis. Besides this form, there are sporadic cases of inflammation of the pia mater of traumatic origin, or due to inflammation of the dura mater; and, lastly, although rarely, cases that must be referred to catching cold, or some other injurious influence acting on the body

Anatomical Appearances.—Inflammation of the dura mater is never spread over large surfaces, but is always more or less circumscribed. At first the inflamed spot appears injected, infiltrated, and relaxed; later it becomes discolored, friable, and is occasionally covered with purulent exudation. The results of pachymeningitis spinalis are permanent thickening of the dura mater, and its adhesion to the bone; more rarely there is perforation of the dura mater by the pus collected between it and the bone, and, as a consequence, diffuse meningitis.

We must regard as remains of chronic inflammation of the arachnoid, milky opacities of that membrane, and cartilaginous or bony plates, about the size of a millet-seed, which have a rough inner and smooth outer surface, and which are generally very numerous, especially in the lower portion of the spinal cord.

Inflammation of the pia mater is usually very extensive. In the acute form we find the pia mater injected, swollen, and relaxed. In the subarachnoid space there is a copious, purulent, flocculent exudation, or else the pia mater and arachnoid are covered with membranous deposits. The spinal medulla is usually pale and bloodless; rarely it is injected, relaxed, or softened. Most cases of so-called hydrorhachis acquisita appear to be due to chronic inflammation of the pia mater. We spoke of opacities and œdema of the cerebral membranes as very frequent *post-mortem* appearances in topers, and referred them to a chronic meningitis; it is also probable that inflammatory disturbance of nutrition of the meninges induces the collections of large amounts of fluid in the subarachnoid space of the spine, when they do not depend on general dropsy or atrophy of the spinal marrow (in which case they are unimportant, and do not cause any symptoms). Moreover, it is difficult to decide, from the tension of the dura mater before it is incised, or by estimating the amount of liquid that escapes after it is incised, whether the amount of subarachnoid fluid is abnormally increased. The more cloudy or bloody the escaping fluid, the more probable it is that the hydrorhachis is due to chronic inflammation of the meninges.

Symptoms and Course.—Inflammation of the dura mater is not accompanied by very prominent or characteristic symptoms when it does not lead to perforation, and, by escape of pus into the subarachnoid space of the spine, induce diffuse meningitis. When patients, who have had an injury of the back, or have caries of the spine, complain of pain in the back, it is difficult to decide whether it is due to inflammation of the dura mater or of the bones and ligaments.

The symptoms of chronic inflammation of the arachnoid, and during the formation of the little plates above described, are perfectly obscure.

Acute inflammation of the pia mater is accompanied by symptoms of severe irritation in the parts supplied by spinal nerves, which are subsequently generally followed by symptoms of paralysis; it is usually distinctly characterized and readily recognized by these symptoms and their sequence. Occasionally after a chill there is fever, and the patients complain of severe pain in the back, which becomes insupportable on motion, and usually on pressure over the spine. It is ordinarily accompanied by pains in the extremities. Both the pain in

the back and the peripheral pain are to be referred to the irritation of the sensory nerves within the spinal canal, from the inflammation of their envelopes. Tonic spasms in the muscles of the back and extremities, causing opisthotonos and contraction of the limbs, are just as constant symptoms as the morbid excitement of the sensory spinal nerves. These tonic spasms, which, as is well known, we may induce artificially in animals, by irritating the spinal medulla with the induced current, usually remit and exacerbate. The exacerbations are not, as in tetanus, induced by irritating any part of the skin, but by movements of the spinal column, a fact which indicates that the tonic spasms in meningitis spinalis are not due to increased reflex excitability, but are direct results of irritation of the motor nerves from the inflammation of their envelopes. The rigidity and tension of the body, which prevent the patient, who is not at first paralyzed, from moving, are from time to time interrupted by convulsive starts. If the respiratory muscles participate in the tetanic rigidity, breathing becomes impossible, and the patient soon dies as a consequence. If the respiratory muscles remain unaffected, there is occasionally a gradual improvement; but more frequently paraplegia is developed, or the fever increases, and the patient succumbs to the paralysis, which advances to the medulla oblongata, or to the exhaustion induced by the fever.

In the chronic form of inflammation of the pia mater, whose symptoms we described with those of hydrorhachis acquisita, since we consider a distinction between them as impracticable, the pain in the back is usually inconsiderable and is easily overlooked. On the other hand, at the onset of the disease, the pain in the extremities is often such a prominent symptom, that the affection is mistaken for peripheral rheumatism. The most important symptoms are those of paralysis, which, commencing in the lower extremities, extend to the bladder, rectum, and subsequently to the upper extremities. The paralysis is usually incomplete at first, and only gradually increases to complete paraplegia; along with it there is usually a feeling of formication and furriness in the lower extremities, the precursor of anæsthesia, which, however, rarely attains a high grade. In some of these cases the paraplegia develops quickly, occasionally in a few days after being preceded only for a short time by pain, which is considered as rheumatic (hydrorhachis rheumatica). The paralysis then often remains stationary at the height it has attained, or may entirely disappear. In other cases the paraplegia develops more slowly and insidiously. In such cases the hope of a permanent decrease of the paralysis is slighter, although the disease almost always shows remarkable variations in its course. Most patients die sooner or later from extension of the paralysis to the medulla oblongata, from bed-sores, or from

catarrh of the bladder. Paraplegia occurs in some other spinal diseases as well as in chronic meningitis. The gait of the patient is not characteristic, and does not differ from the gait in other forms of paraplegia. The old belief that the symptoms of paralysis, due to a collection of fluid in the spinal canal, grew worse when the body was upright and less when it was horizontal, from the fluid being distributed more evenly, was purely theoretical and not derived from direct observation. The most important points, in distinguishing chronic meningitis and hydrorhachis acquisita from other diseases of the spinal cord, are the symptoms of irritation, particularly the painful sensations which precede the paralysis; also the gradual advance of the paralysis from below upward, which does not occur in the disease limited to certain points, and particularly the varying course of the disease, the exacerbations and remissions, which do not occur in diseases destroying the spinal medulla.

Treatment.—Acute spinal meningitis requires energetic antiphlogistic treatment, particularly the application of leeches and wet cups to both sides of the spine. If the disease be of traumatic origin, we should at the same time use cold to the back by means of an ice-bladder, or the frozen compresses before mentioned. In very recent cases, as there is danger in delay, and not much can be lost, we may employ the much-lauded frictions with mercurial ointment, and give calomel internally. If the acute stage passes off and the disease is protracted, we may apply flying-blisters to both sides of the spine; commencing with them at the neck, we gradually descend to the sacrum, and then begin again at the neck. In meningitis, flying-blisters appear to be more efficient than moxæ and the hot iron, which deserve the preference in diseases of the vertebræ, and in inflammations of the spinal medulla limited to certain points. In protracted cases, or at the commencement of those running a chronic course, cold plunge-baths and douches, and particularly continued warm baths, are very serviceable. The reputation of these remedies, in paraplegia generally, depends chiefly on their results in chronic meningitis spinalis, which is the most amenable to treatment of the diseases of the spinal medulla and membranes. Such patients are also benefited by the bath-treatment at Wildbad and other similar thermal springs.

CHAPTER IV.

INFLAMMATION OF THE SPINAL MARROW—MYELITIS.

In the present chapter we discuss also softening and hardening of the spinal medulla, myelomalacia, and myelosclerosis, since we shall consider these degenerations (except where the softening is due to

hæmorrhage and œdema, as already described) as results or forms of myelitis until we shall have learned some other mode of origin for them, as we have for the analogous changes in the brain.

Etiology.—Myelitis induces the same structural changes as encephalitis does. In it there is no abundant interstitial exudation, but the nerve-elements undergo inflammatory disturbances of nutrition, and finally break down, just as the ganglion-cells and nerve-filaments of the brain do in encephalitis.

Apart from the cases due to propagation of inflammation from the vertebræ to the medulla, myelitis must be regarded as a rare disease. But this extension occurs quite frequently. Most paraplegias which come on during vertebral disease are not results of the curvature of the spine, but of inflammation extending from the vertebræ to the membranes, and thence to the medulla. This view is supported by the fact that in vertebral disease there is frequently paralysis before the spine is curved, and, on the other hand, great curvature often exists for years without symptoms of paralysis, till these at last accompany the deformity without any increase of the curvature, but usually after pain in the back. More rarely, wounds and contusions of the spinal column, or the development of syphilitic exostoses, induce myelitis. Occasionally the disease develops about neoplasia and extravasations of blood. Lastly, sexual excesses, excessive straining, catching cold, suppression of the perspiration of the feet, etc., are given as causes of myelitis. We do not know whether in such cases the disease is really due to these causes or to other unknown influences. Patients with paraplegia, besides their other misfortunes, are usually subjected to the unfounded suspicion that they have brought on their disease by dissolute habits.

Anatomical Appearances.—Myelitis is sometimes confined to circumscribed spots, and then, usually starting from the gray substance, it attacks the whole thickness of the medulla; sometimes, as "central softening," it extends widely through the gray substance, and then affects the white substance but little.

In the *circumscribed spots* we find the spinal medulla swollen in recent cases. If we cut into it, a more or less consistent red pulp (red softening) rises above the cut surface. In older cases, the color of the pulpy soft spot becomes more brown or yellow (yellow softening), from change of the hæmatin and fatty degeneration of the broken-down nerve-elements. Far more rarely than in the brain, the inflamed spot in the spinal medulla is converted into an abscess by the extensive formation of pus-cells. The inflammation in the meninges usually extends beyond that of the medulla. From disintegration and reabsorption of the disintegrated elements in circumscribed myelitis a

cavity, filled with serum and traversed by delicate connective tissue, may be formed in the spinal medulla. In other cases there is induration from connective-tissue proliferation. These sclerosed spots, which are usually somewhat retracted and colored yellow by pigment, form analogues to the yellow plates, which we found as remains of peripheral encephalitis.

In the second (central) form of myelitis we find the medulla but little swollen at first. On section, the contours of the gray substance appear lost; it is somewhat darker in color, reddened, and less consistent. In older and typical cases the spinal medulla is decidedly swollen, and in its centre we find a thin reddish, rusty, or yellow pulp. In this form, also, the tissue-elements, which have broken down to a fine detritus, are sometimes reabsorbed, and fluid exuded in their place, so that, at last, in the axis of the spinal medulla, there is a cavity filled with serum, and surrounded by delicate connective tissue, or traversed by a framework of the same.

Symptoms and Course.—Since acute myelitis is always accompanied by meningitis, it is also accompanied by the symptoms of spinal meningitis described in the last chapter, and we can only decide, from certain modifications of the symptoms there described, that the medulla itself, as well as the meninges, is inflamed. In the commencement of the disease there is usually severe fever and more or less extensive pain in the back and extremities, tetanic rigidity of the muscles of the back and nape of the neck, contractions of the extremities, alternating with convulsive attacks, and where the respiratory muscles participate in the tetanic tension there is great dyspnœa. The more circumscribed the pain in the back, the more distinctly the peripheral pains and spasms are limited to the parts supplied by nerves from a certain portion of the medulla; but especially the earlier and more completely paraplegia follows these symptoms of irritation, the more probable it becomes that the spinal medulla itself is inflamed. In very malignant cases these symptoms may get the upper hand very quickly, and, even in the first days of the disease, the patient may die from disturbance of the respiration. In other cases the storm passes over, but a paraplegia remains, which hardly ever recovers. Cases running the above course are rare, and are almost always of traumatic origin, or result from perforation of an abscess into the vertebral canal.

Chronic myelitis, also, is generally preceded by symptoms of irritation; but, as the participation of the meninges in the tedious inflammation of the spinal medulla is slighter and less extensive, they are usually limited to vague pains, formication, momentary twitching, or painful contractions of the extremities. At the same time many patients complain of a dull pain in the part of the spine corresponding

to the point of inflammation, which is increased by pressure on the spinous processes, but not by movements of the spinal column, which is an important point in the diagnosis between myelitis and meningitis spinalis. This pain is sometimes accompanied by the feeling of a cord tied firmly around the waist. In other cases there is no spontaneous pain, but the corresponding vertebræ are sensitive to pressure. If we pass a sponge, previously dipped in hot water, along the spine, the part at the seat of inflammation is generally more sensitive than elsewhere. These symptoms, which are usually little thought of, are accompanied by a heaviness and helplessness in the lower part of the body, which sooner or later become perfect paraplegia. The higher up the seat of the inflammation, the more extensive the paralysis. If the lumbar region be diseased, the lower extremities are paralyzed; if the thoracic region be affected, the sphincters participate in the paralysis; if the cervical region suffer, the paralysis extends to the upper extremities and corresponding respiratory muscles; with the paraplegia there is generally also anæsthesia of the lower part of the body, but this rarely extends to entire loss of sensitiveness to irritation. While the paraplegia and anæsthesia, slowly increasing, attain a high grade, the affected muscles are still occasionally attacked by spasmodic twitchings, and, if the anæsthesia be incomplete, by painful contractions. This symptom is readily explained by the irritation induced in the motor nerves of the extremities from the progress of the inflammation, even after their connection with the central filaments is lost. In cases where the connection between the motor nerves and central filaments is completely broken, so that no muscular contractions are induced by the will, it is not at all impossible for an excitement to pass from the sensory to the motor filaments, and for reflex movements to occur. On the contrary, we often see the reflex excitability increased in the parts of the spine below the interruption. This pathological experience exactly corresponds with experiment. It is well known that, in decapitated animals, reflex movements occur more readily than in those whose motor nerves are under the influence of the brain. In Greifswald I saw a young woman, who was paraplegic as a result of vertebral disease, in whom the reflex symptoms in the paralyzed part were so severe that the slightest touch on the skin of the lower extremities caused their muscles on both sides to contract spasmodically. It was very interesting to observe that, in this case, when, contrary to all expectation, the paralysis improved, and the patient was able to make voluntary movements of her extremities, the inclination to reflex symptoms entirely disappeared. The course and results of chronic myelitis vary. The disease may run on for years; frequently it advances to a certain point, and remains stationary. If

such patients belong to the educated classes, or to the mechanics proper, they often continue their occupation in spite of their paraplegia. Those cases of paraplegia, where improvement and recovery take place, as I before said, appear to belong to meningitis spinalis, not to myelitis, as it is very improbable that nerve-elements, which have been extensively destroyed, can be regenerated; the favorable course of incised wounds of the nerves does not disprove this assertion. In chronic myelitis, death most frequently results after the patient has been confined to bed a long while by the increasing paralysis. The consequent bed-sores, or the cystitis caused by stagnation of the urine, generally form the terminal symptoms, if the patient do not sooner die of tuberculosis or intercurrent diseases.

Treatment.—The treatment of myelitis promises little. It is to be conducted on the principles laid down for meningitis spinalis. Only, instead of flying blisters, it is well to apply moxæ or the hot iron near the supposed seat of inflammation.

CHAPTER V.

GROWTHS AND PARASITES OF THE SPINAL MEDULLA AND ITS MEMBRANES.

Except the cartilaginous and bony plates of the arachnoid, which we have already described, growths rarely occur in the spinal canal. Carcinomata, which are usually of the medullary variety, either form primarily in the spinal medulla or dura mater, or extend from the vertebræ to the meninges and medulla. When they grow considerably, they may at last fill the spinal canal, as the medulla atrophies from pressure, or is transformed into cancer substance. Occasionally, after destruction of the vertebræ, they have been seen to spread outwardly, so as to lie just under the skin. Only a few cases of sarcoma and glioma have been observed in the spinal canal. They almost always started from the inner wall of the dura mater, and rarely attained any considerable size. Tubercles in the medulla itself only occur when there is advanced tuberculosis of other organs. Just as in the brain, they usually form yellow nodules, the size of a pea or hazel-nut. They are usually in the cervical or lumbar regions. Somewhat more frequently we meet tuberculous degeneration of the dura mater, under the form of so-called infiltrated tuberculosis, along with tuberculous caries of the vertebræ. Cysticerci and echinococci have also been very rarely found in the spinal canal. The latter had either developed between the membranes, or an echinococcus sac, near the spinal

canal, had penetrated into the canal, after destroying the vertebræ or their processes.

Tumors in the spinal canal interrupt the communication between the brain and peripheral nerves; hence they cause paraplegia and anæsthesia of the lower part of the body. According as they simply induce atrophy of the spinal medulla by pressure, or cause inflammation of it by the irritation of the parts about them, the paraplegia and anæsthesia are preceded by moderate or by very severe symptoms of irritation. Of course, the seat of the growth modifies the extent of the symptoms. The fact that, in tumors, there is usually less pain in the back, but more peripheral neuralgia preceding the paralysis, and that the paralysis often did not begin at the same time on both sides, but gradually extended from one side to the other, is not absolutely decisive in the differential diagnosis between tumors of the spinal medulla and chronic myelitis. We can only make an absolute diagnosis in those cases where the cancer extends from the spinal marrow outward. The development of carcinoma, tubercles, or parasites in other organs, at least justifies the suspicion of a similar disease in the spinal canal being the cause of a slowly-progressing paraplegia. Treatment is entirely powerless against all tumors of the spinal medulla.

CHAPTER VI.

HYDRORHACHIS CONGENITA—SPINA BIFIDA.

Hydrorhachis congenita is divided into internal and external. The former depends on a collection of serum in the dilated fœtal central canal. It causes atrophy or entire destruction of the spinal medulla (amyelia) by pressure, or else splits it more or less completely. Hydrorhachis externa consists in an abnormal collection of water in the subarachnoid space. In both forms the vertebral canal may either remain closed (hydrorhachis incolumis), or there is, at the same time, a more or less extensive opening of the canal (hydrorhachis congenita dehiscens).

In spina bifida we find a sac filled with serum, and covered by the spinal membranes on the spinal column, which communicates with the spinal canal, as a result of rudimentary formation of one or several vertebral processes. Such tumors are usually located in the sacral or lumbar regions, more rarely in the cervical or dorsal. Their size varies from that of a walnut to a child's head. The skin covering them is sometimes normal, sometimes thinned; occasionally, at the summit, it has entirely disappeared; then the sac is exposed, the place appears excoriated, and is occasionally covered with pus and granulations.

When the spina bifida results from hydrorhachis externa, the wall of the sac consists of the arachnoid and dura mater; the latter is occasionally thinned or perforated, and then the wall consists of the arachnoid alone. If, on the other hand, the spina bifida result from hydrorhachis interna, the pia mater also assists in the formation of the wall. The contents of the sac are pure serum, of the same constitution as the cerebro-spinal fluid. When the spina bifida has resulted from hydrorhachis interna, the spinal medulla is either altogether absent, or is undeveloped. On the other hand, if it be due to hydrorhachis externa, the medulla may be perfectly normal; but occasionally, even in such cases, it is defective (*Förster*).

The pathogeny and etiology of hydrorhachis congenita are obscure. The collection of water is probably the primary disease, the incomplete formation of the spinal canal the secondary.

The symptoms of hydrorhachis, complicated with partial spina bifida—of which alone we shall speak, as, in all cases combined with great defect of the spinal medulla and extensive opening of the canal, the children die before birth, or very soon after—consist chiefly of the objective signs due to the above-described tumor. There is usually distinct fluctuation in it. At its base the edges of the bone may be felt. It increases on expiration, still more on crying and straining, and diminishes on inspiration. Occasionally it may be replaced, but attacks of loss of consciousness and general convulsions are readily induced by these attempts. In some cases the innervation of the lower extremities, bladder, and rectum, is normal; in others, especially where the lower part of the medulla is defective, or has disappeared, the lower extremities, bladder, and rectum, are paralyzed. The tumor usually increases rapidly in size and tension soon after birth. If the skin reddens, becomes thin, and finally perforated, death, preceded by convulsions and subsequent sopor, generally quickly follows the perforation. But, even where perforation does not take place, most of the children die early of general marasmus, and it is very exceptional for patients with spina bifida to attain or pass the age of puberty.

Operative procedures are to be abstained from when complete paralysis and decided emaciation of the lower extremities indicate that the spinal medulla is incompletely developed. In other cases we may attempt careful compression, and, where this fails, proceed to the operations described for spina bifida in the text-books on surgery.

CHAPTER VII.

CONSUMPTION OF THE SPINAL CORD—TABES DORSUALIS—ATAXIE LOCOMOTRICE PROGRESSIVE (*Duchenne*)—GRAY DEGENERATION OF THE POSTERIOR COLUMNS OF THE SPINAL CORD (*Leyden*)—LOCOMOTOR ATAXY.

Tabes dorsualis, which was discovered and described by *Duchenne* as ataxie locomotrice progressive, long after it was generally known in Germany from the classical description of *Romberg*, has recently excited much discussion. The views regarding the significance of the symptoms especially vary. According to the plan of my book, I can only criticise the views of others, so far as is necessary to support my own. The following description is, in the main, the same as was given in the dissertation of my former assistant, Dr. *Ernst Spaeth*, which was written under my supervision, and which has not, by any means, received the attention it deserved. (The dissertation of Dr. *Spaeth* was published under the title of "Beiträge Zur Lehre von der Tabes dorsualis," at Tübingen, 1864, by Aug. Ludwig.)[1]

Etiology.—In some few cases of decided tabes dorsualis, on autopsy we find no palpable changes in the spinal medulla. This "negative appearance" does not by any means prove that the spinal medulla is normal, but only that its functions may be impaired by molecular changes that escape observation, just as they are by the coarser changes found in other cases. The latter consist in a peculiar degeneration and atrophy affecting particularly the posterior columns and the posterior roots, but occasionally the gray substance also and other columns. In some cases there is no doubt that the degeneration in question is the result of inflammation of the spinal medulla, but in other cases no inflammatory origin can be made out.

The predisposition to tabes dorsualis is occasionally hereditary, as is shown by a number of cases, where several brothers and sisters were attacked by the disease. On the other hand, I only know of two cases where it was inherited, that is, where the parents of the patient had tabes dorsualis. Children are not inclined to tabes, persons of mature age are most so, while in the aged the tendency almost disappears. Men are far more disposed to it than women.

The exciting causes are almost always said to be—1. Venereal excess. The great physiologist *Johannes Müller*, usually very careful and discreet in his assertions, says directly that tabes dorsualis comes *only* from venereal excess. Besides their other sufferings and misfortunes, these poor patients are made to bear the accusation which in many cases at least is unjust, that they have themselves to blame for

their disease. There is no doubt that many of the patients attacked have been addicted to excess in venery, but there is just as little doubt that many patients with tabes dorsualis have led an exemplary life, while others who have been unbounded in their excesses escape the disease.

From the great frequency of lewd habits, and from the difficulty of deciding the amount in any given case, I consider the dependence of tabes on venereal excess as not yet proved. 2. On the other hand, there appears to me no doubt of the influence of *catching cold* and *bodily fatigue*, especially when they act together, in inducing tabes dorsualis. The disease occurs with remarkable frequency among those who, wearied and heated by severe marches or other bodily exertions, have stretched themselves on the damp earth to rest. 3. Many patients ascribe their disease to suppressed perspiration of the feet. We cannot generally lay much weight on these accounts, for the laity give suppression of habitual perspiration as a very common cause of disease. Doubtless in most cases where the occurrence of a disease is accompanied by the absence of habitual perspiration of the feet, it is because the latter has ceased with the appearance of the disease. But I think it is going too far to deny the possibility of a genetic connection between the arrest of perspiration of the feet and tabes dorsualis. At all events, many patients with tabes have previously suffered much from perspiring feet. 4. Lastly, the disease appears occasionally to be of syphilitic origin, a supposition chiefly supported by the fact that some tabes patients who had previously suffered from syphilis were benefited by an antisyphilitic treatment.

Anatomical Appearances.—The usual appearance of the spinal medulla and its membranes, in decided cases of tabes dorsualis, is as follows: The dura mater is either unchanged or its posterior half is slightly thickened, the arachnoid is moderately opaque, if the medulla be atrophied the fluid in the subarachnoid space is increased; on the posterior surface the pia mater is constantly thickened, clouded, and more or less adherent to the posterior columns.

In the early stages, where no decrease of volume of the spinal medulla is as yet observable, there is a peculiar degeneration of the posterior columns; this always begins in the immediate vicinity of the posterior fissure, close under the pia mater, and thence spreads toward the sides and the gray commissure; the diseased part always retains the shape of a wedge, with the base directed toward the pia mater. The degeneration consists in a transformation of the white substance of the posterior columns into a gray or grayish-red, half-translucent, soft mass. On microscopical examination of the latter we only find a few nerve-filaments, partly in various stages of atrophy; most of them have completely disappeared, and between the atrophied cells there is a richly

nucleated connective-tissue substance, corresponding to the ordinary neuroglia and resulting from its proliferation, in which we find only a few granular cells, molecular fatty masses, and numerous corpora amylacea. The vessels passing out from the pia mater have a greatly-thickened adventitia, and thus cause the firm adherence of the pia mater to the surface of the medulla. Corresponding to the extent of the degeneration in the posterior columns, the posterior roots of the spinal nerves are atrophied. They resemble thin, vascular, translucent connective-tissue cords, far more than they do nerves with medulla. This is especially true of the posterior roots of the cauda equina. The anterior roots of all the nerves, cauda equina included, are normal, corresponding to the general nutritive condition of the spinal medulla.

In the later stages the degenerated gray parts of the medulla shrink greatly. They become a hard tissue, and are less transparent. If the degeneration be limited to the posterior columns, the medulla acquires a cylindrical form. The points of exit of the posterior roots are approximated. If, on the other hand, the degeneration extend to the posterior parts of the lateral columns, the medulla shrinks more in an antero-posterior direction, and we may readily receive the impression that it has become broader. The neuroglia, which in the early stages of the degeneration corresponds exactly with the normal neuroglia, during the shrinking acquires the appearance of a fine filamentary substance. A secondary atrophy and induration succeed the growth of the neuroglia.[2]

Symptoms and Course.—Although *Duchenne* is to be reproached for ignorance of *Romberg's* works, or else for ignoring them, when he published his first writings on *ataxie locomotrice progressive*, we cannot deny that he has done much for the correct interpretation of the symptoms of tabes dorsualis. He originated the doctrine, at present almost generally adopted, that in tabes there is not paralysis, but disturbed coördination of muscular movements. To use our muscles properly, it is not enough that we can contract each muscle, we must also be able to cause a harmonious action of all the muscles participating in any motion. Acts apparently the most simple fail or are clumsily performed, if any of the muscles participating in them be contracted too much or too little, too quickly or too slowly; or if the antagonists be not relaxed just enough or exactly at the right moment. This power of causing the muscles to act properly together, or, as it is usually called, the power of coördination, is much impaired in patients with tabes; and the symptoms resulting from this anomaly are peculiarly characteristic of the disease. But, along with this, there is almost always a decided diminution of the cutaneous and muscular sensibility, which *Romberg* has excellently described: "The floor is no longer distinctly

felt; the foot seems to rest on wool, soft sand, or on a bladder filled with water. The horseman no longer feels the resistance of the stirrup, and has the strap shortened .... If the patient do not *see* his movements, they will be still more uncertain; if, while erect, he closes his eyes, he immediately begins to sway about and totter. If his eyes be closed while in the horizontal position, he cannot tell the location of his limbs; he cannot say whether the right foot be crossed over the left, or the reverse."

If, as some celebrated authorities assert, the posterior spinal columns have the function of coördinating the movements, the anatomical changes found on autopsy of tabes patients fully explain the symptoms observed during life. We have said that the degeneration and atrophy of the spinal cord constantly start from the posterior columns, and that the posterior sensory roots almost always participate in the degeneration. *Leyden*, who does not believe in a peculiar power governing the coördination of movement and its location in the posterior spinal columns, has advanced a theory for the explanation of the disturbances of coördination in tabes dorsualis, which, at first sight, is very enticing. He explains the loss of power of coördination as due solely to the diminished cutaneous and muscular sensibility. We must, indeed, admit that *Longet* is correct in saying that a person, who has lost the perception of his actions, who cannot judge of the position of his limbs, who does not even know whether they are present, and, lastly, who does not feel the floor under his feet, cannot walk erect, preserve his equilibrium, and move his limbs with certainty and accordance. Moreover, the peculiarities of the anomalies of movement observed in tabes patients, the energetic lifting of the foot and its passive fall when walking, the spasmodic, uncertain movements, their shooting beyond the mark, all give the impression that the patient is so awkward because he does not know what he has done till he has done too much. Lastly, the fact that the helplessness of the patients is greatly increased when they close the eyes, and cannot control their movements by the sight, is favorable to the view that the anomalies of movement in tabes dorsualis result from diminution of the cutaneous and muscular sensibility. Nevertheless, *Leyden's* theory is false. Its correctness is opposed by the following facts: first, that in many patients the disturbances of sensibility are in marked disproportion to the impairment of coördination of movement; second, that in persons whose cutaneous and muscular sensibility is far more diminished than is the case in any tabes patients, there is often no indication of disturbance of coördination. In *Spaeth's* work there is a full history of a peasant, from Wurmlingen, who is known to all my students, as I show him in my clinic almost every half year. This interesting pa-

tient has a very marked and extensive anæsthesia of the skin and muscles. He is insensible to the severest injuries. He does not know if he bathes in warm or cold water. On loading his extremities with a weight of twenty-five pounds, he perceives no difference of pressure. On being requested to estimate weights, by lifting them, he cannot distinguish between weights of one pound and a hundred pounds. When his eyes are closed, he cannot tell whether his limbs are flexed or straightened to the greatest possible extent by strong electric currents. When standing erect or sitting up, if he closes the eyes he immediately falls. He perceives the resistance of his bed so little that, when the light is extinguished at night, he feels as if he were swimming in the air. But this patient has no marked disturbance of coördinative power; he does not in the least remind us of a tabes patient; as long as it is light he walks very well, although carefully; he travels on foot, without a stick, the mile between Wurmlingen and Tübingen. A single such case is enough to prove that the disturbance of coördination of the tabes patient does not depend, certainly not solely, on the diminished sensibility, but that it exists along with the latter.

In many cases the above symptoms are accompanied by disturbances of the excretion of urine. Most patients are obliged to attend to the call to urinate as quickly as possible, as they can only stand it a few moments, and hence, when their means allow it, they buy urinals, which they wear in their trousers during the day. I think this symptom arises because the patient does not perceive the fulness of the bladder, and the desire to urinate does not occur till a few drops are pressed out of the bladder into the urethra. Far more rarely than this incomplete enuresis we find retention of urine, and it becomes necessary to draw it off with the catheter. In such cases there is probably paralysis of the bladder, from its having been distended too much or too long.

It is asserted that, at the commencement of the disease, sexual desire is usually increased but the energy and duration of the erections lessened, as well as that, in the later stages, the virile power is entirely lost. The latter assertion alone is certain.

We must also mention the paresis of the oculo-motor and abducens, which occasionally occurs in tabes, which is shown by diplopia, more rarely by strabismus and ptosis of the upper lid; and, lastly, the amaurosis and psychical disturbances which are sometimes met with. There is no doubt that these symptoms are due to the disease advancing to the cerebral filaments, but hitherto autopsy has failed to show what course this advance takes. Atrophy of the optic nerve was only found in some of the few cases where there was disturbance of vision; but even here the atrophy only extended to the corpora quadrigemina, and could not be followed further.

After having described and analyzed the most important symptoms, I shall attempt to give a general description of tabes dorsualis, and picture the most frequent course of the disease as briefly as possible.

In many patients, the characteristic disturbances of coördination, and the decrease of cutaneous and muscular sensibility are preceded for a long time, even for years, by attacks of severe tearing pain in the lower half of the trunk, and in the lower extremities; these are generally considered as rheumatic. In other patients, on the contrary, the first complaints are that the lower extremities become fatigued very easily and very soon. Persons accustomed to walking notice that they tire sooner and more easily than formerly. These symptoms, which are not usually very suspicious, either to the patient or physician, may precede the decided symptoms of tabes for a long time. But this difference in the initial symptoms is not so marked or decided as it seems to be on superficial examination. The attacks of pain are unmistakably neuralgic, and depend on morbid excitement of the posterior roots, while the tendency to fatigue depends on their hyperæsthesia—that is, on their morbidly-increased excitability. Under physiological circumstances, the feeling of fatigue depends on the amount of work done by the muscles. The state of the muscles resulting from overwork is perceived through the sensory muscular nerves. If the excitability of the posterior roots be increased, slight exertion of the muscles will produce the same effect, which would otherwise only be induced by far greater exertion. Hence the tendency to fatigue in tabes is perfectly analogous to the increased sensitiveness, at the commencement of certain brain-diseases, to light, sound, and other irritations, which are not generally unpleasant. The tearing-pain and tendency to fatigue in the lower extremities are not recognized to be serious and threatening until they are accompanied by other disturbances of sensibility, such as formication, a feeling of furriness, numbness, and the sensation of a ligature around the abdomen. Gradually the gait becomes uncertain and awkward; at first this is so only in the dark, so that the patients prefer remaining at home in the evening; afterward it is the same in day-time. The feet are lifted too high, and thrown forward and outward, and then brought heavily down on the floor. If the patient closes his eyes while standing erect, he begins to totter, and, unless supported, falls to the ground. And, even at this time, he must usually be on his guard when the desire to urinate seizes him, so that he may reach a convenient place in season. After a time, walking becomes impossible, even with the aid of a stick or crutches, although, when the body is fixed, the patient can generally make uncomplicated movements of the extremities with nearly normal force. The same uncertainty and awkwardness come on in the upper ex-

tremities much later than in the lower. Then, when eating or drinking, the patients shake out the contents of the spoon or glass, they can no longer dress without aid, especially the buttons trouble them; finally, they cannot write, knit, or do any other work. The enuresis now often attains such a grade that the patient voids his urine in bed. If, in consequence of this, a constant moisture of the parts be added to the patient's indistinct perception of the irregularities of the bed and his helplessness in changing his position, we may readily have bed-sores.

The course of the disease is always tedious. We see most patients drag on for years in a miserable state, the objects of pity, which is not unfrequently pharisaical. Occasionally the disease remains stationary; in other cases it appears to improve temporarily. Perfect cures are certainly very rare.

Nutrition is frequently not impaired till late; the lower extremities, the nates, and muscles of the back emaciate first, so that the spinous processes project. It is not till toward the end of the disease that the emaciation extends to the rest of the body. Death generally results at last from the increase of the bed-sores, from severe cystitis, from pulmonary consumption, or from intercurrent diseases.[3]

Treatment.—*Romberg's* assertion, that there is no hope of cure for a tabes patient, that they are all doomed, contrasts strongly with the views of *Remak*, as published by *Cyon*, according to which the former is said to claim that his treatment was successful in the majority of cases, and that, therefore, he cannot be reproached for not having autopsies on his cases. The truth probably lies between the two. Of late, few authorities regard the treatment of tabes so hopeless as *Romberg*. But very few would agree with *Remak*, *Benedikt*, and others, in saying that tabes is a disease where very favorable results can be attained by treatment.

We may hope for the best results when there is a suspicion that the disease is of syphilitic origin. In such cases an antisyphilitic treatment should be instituted, on the plan to be described hereafter.

The more probable it is that the disease has resulted from taking cold, the more acute its occurrence, the more severe the pains in the lower extremities which preceded the symptoms of disturbance of coördination and diminished sensibility, the more probable is it that the disease is of inflammatory or congestive origin, and the stronger the indications to begin the treatment with the local abstraction of blood by leeches, and derivation to the skin by blisters along the spine. Subsequently we may order for such patients the waters of Wildbad, Gastein, Ragaz, Pfäffers, Töplitz, etc. There is no doubt that tabes patients have been decidedly benefited by the treatment at these places. On the other hand, I must warn against the careless employ-

ment of the cold-water treatment, which, especially in the form of cold douches to the back, has not come up to the expectations entertained of it at the time it was fashionable, and the water-cure establishments are now rarely patronized by tabes patients, who, for a time, all rushed to them for treatment. In very recent cases only, sweating in moist cloths and a subsequent short bath appear to be beneficial.

If it be not a favorable time of year to send the patient to Wildbad, etc., or if these mineral waters do not seem indicated, or if they have done no good, we should try the administration of nitrate of silver as recommended by *Wunderlich*. Nitrate of silver has long held the reputation of being one of the most effective nervines; and it is not at all impossible or improbable that it exercises a modifying influence on the nutrition of the nervous system; it is not these reasons, however, but the fact that reliable observers have found some benefit from the employment of the remedy in tabes dorsualis, that induce me to use it in most cases of the disease. My experience of the efficacy of the remedy, which I have given in a large number of cases, in doses gradually increasing to half a grain daily as advised by *Wunderlich*, has not been particularly favorable. It is true that most of my patients at the *clinic* have praised their state a time after using the remedy, but I suspect that many did so to induce me to keep them longer at the hospital. In other cases, the patients actually seemed to move forward more nimbly; but here also it was possible that either greater confidence or increased attention of the patient, with the view of showing me improvement, had a favorable effect on the attempts to walk. I shall continue for a time to prescribe nitrate of silver to my tabes patients.

My experience of the constant current of electricity is about the same as it is in regard to nitrate of silver. I have no reason to doubt that *Remak*, *Benedikt*, and others, have had some success with the constant current which I consider to be a very active remedy, but thus far I cannot claim any success from its use in tabes, although I have had an excellent apparatus in my clinic these four years, and have treated all my tabes cases with it in the manner advised by *Remak* This want of success will not prevent my continuing the use of galvanism in tabes for a time; and, instead of letting the current act on the spinal column as hitherto, I shall try the current through the spinal nerves, as advised by *Benedikt*, not because I attach much importance to the theories and the indications he gives for the treatment of different diseases, with the current through the roots of the spinal nerves (Ruckenmarks-Wurzelströmen), through the spinal nerves (Ruckenmarks-Nervenströmen), etc., but simply because I have no reason to doubt what he says on the subject.

ADDITIONS TO THE REVISED EDITION OF 1880.

SECTION II.—DISEASES OF THE SPINAL MARROW AND ITS MEMBRANES.

1.—P. 302.

Some diseases may affect the entire cross-section of the spine unequally, while others are limited to certain parts. In the latter only certain physiological functions are affected, so that their symptoms must differ. Infantile spinal paralysis and progressive muscular atrophy are affections of certain parts of the anterior portion of the medulla spinalis, while in locomotor ataxy the posterior part is affected.

2.—P. 304.

It is doubtful whether the disease starts from the nerve-filaments or from the intervening substance ; the fact of the process being generally limited to the posterior columns favors the former view. The vessels are sometimes unchanged, at others their adventitia is thickened and fatty, their calibre diminished or closed.

Some observers think the affection is due to disease of certain limited portions of the posterior roots—two lateral, thin, gray striæ, corresponding to the intermedullary extensions which *Kölliker* terms inner root-filaments. According to *Charcot*, sclerosis of these lateral striæ is the only constant anatomical change. The sclerosis may spread from the posterior columns to other parts, as the posterior nerve-roots, or even to different nerves, as the sciatic, crural, and brachial, but especially to some cerebral nerves, as the optic, oculo-motor, trochlear, and hypoglossal. It may also extend to the posterior horns of the gray substance, and to the lateral columns, or to the gray anterior horns ; in which cases, of course, the symptoms will be varied. The disease may begin at any point of the spine, but generally spreads in the long axis ; usually it begins in the lumbo-dorsal region, and spreads along the lateral striæ of the posterior columns to the cervical region, or even to the cerebral nerves and brain, as is shown by psychical changes and depression. On the other hand, primary cerebral disease may be followed by tabes, and it has been asserted that the progressive paralysis of the insane sometimes chiefly affects the posterior roots.

3.—P. 308.

In many tabes patients, sometimes as the first symptom, there is impairment of vision, beginning as concentric limitation of the visual field in one eye, or sometimes as achromatopsia, gradually grow-

ing worse and ending in double amaurosis, with gray degeneration of the optic nerve. Other cerebral nerves are more rarely affected, but there may be anæsthesia of the trigeminus, impairment of hearing, deglutition, or speech, and periodical rapidity, irregularity, or dicrotism of pulse due to the vagus. Implication of the sympathetic is often shown by inequality of the pupils. On the side of the contracted pupil there may be signs of vaso-motor paralysis, such as red cheeks, chemosis, and increased temperature. Among the early symptoms there may be a painless swelling of the joints, most frequently of the knee, then in the shoulder, elbow, etc. *Charcot* regards this as allied to arthritis deformans, and refers it to implication of the gray anterior horns. In a case recently observed, the anterior horns were intact, but the spinal ganglia were very large and perceptibly changed.

The neuralgias are due to inflammatory irritation of the posterior roots of the nerves; there may be formication, tearing, contracting, and especially shooting pains, which may recur weekly or monthly, and are worse at night; they are rarely entirely absent. The period of pain varies in duration; in incomplete cases the latter characteristic symptoms may never occur. As the disease progresses, the pains, which might readily be regarded as rheumatic or neuralgic, spread from the legs to the body, and the sensation of a girdle about the body is noticed; there may be painful contraction of the rectum or bladder, or incomplete erections and pollutions. Later there is neuralgia of other nerves in upper parts of the body, arms, etc.; but there is rarely spinal pain or tenderness. An occasional early symptom is an attack of gastralgia and obstinate vomiting, lasting several days; these attacks often accompany the shooting pains, and seem due to irritation of the sensitive gastric filaments of the vagus.

Perfect cure of tabes is only possible when the sclerosis is but slightly advanced, and even then is probably very rare; though *Benedikt* asserts the contrary, claiming that mild cases are often mistaken for other diseases. Incomplete cures are more common, the progress of the symptoms being arrested and life made endurable. But most cases progress, though some symptoms improve temporarily. As the disease advances, the upper limbs may be affected; formication and neuralgic pains in the arms and fingers precede uncertainty of the grasp in writing, etc., till the patient becomes entirely helpless. Exceptionally, the arms may be first affected; and when the prominent symptoms indicate the part affected to be high in the spine or in the cerebral nerves, a cervical or basilar tabes has been spoken of; but the symptoms are

usually so combined that these regional forms cannot be distinguished.

Although one of the most tedious of diseases, tabes usually shortens life. It may continue five or ten years, or longer ; but the symptoms, especially the motor disturbances, may progress rapidly and death occur early. As the disease advances, the patient may attain a pitiable condition ; the nutrition of the body, which has been good, may suffer, the legs emaciate, the feet become œdematous ; the previous irritable weakness of the sexual function gives way to entire impotence ; urination and defecation are impaired, sensibility blunted, and vision lost ; more especially the power of motion is lost from ataxia and paraplegia. Death is rarely directly due to paralysis of respiratory muscles, and not so often to bed-sores and purulent cystitis as it is in spinal paralysis ; but more frequently it is due to consumption, pneumonia, bronchitis, etc.

In well-developed typical cases diagnosis is easy. The periodical lancinating pains in the legs or arms, quick fatigue, gastralgia, paralysis of ocular muscles, disturbance of vision, and ophthalmoscopic appearances, and especially the ataxia of motion, give the disease a decided character. Diseases of the cerebellum also induce an uncertain tottering gait when the eyes are closed, and dizziness ; but there are usually other symptoms, occipital pain, occasional vomiting, epileptic or tonic spasms, hemiplegia, etc.; while the lancinating pains, ready fatigue, girdle feeling, and anæsthesia are absent. Amblyopia and amaurosis occur from tumors of the brain as well as from tabes ; but the ophthalmoscope may show whether the papilla indicates tabes or the optic neuritis and choked disk due to tumors. Electro-muscular contractility gives no certainty to diagnosis, as the excitability of the muscles may be increased, normal, or lessened ; although in the first stages it is generally increased, while in other spinal diseases it is apt to be lessened very early. When incompletely developed, tabes may be mistaken for neuralgic, rheumatic, hypochondriac, hysterical, or syphilitic affections. Errors may also occur where the posterior columns are implicated in diseases of other parts of the spinal cord.

SECTION III.

DISEASES OF THE PERIPHERAL NERVES.

CHAPTER I.

INFLAMMATION OF THE NERVES—NEURITIS.

Etiology.—Inflammatory disturbances of nutrition rarely occur in the peripheral nerves. They sometimes affect the nerve-filaments, sometimes the neurilemma. The first form ranks among parenchymatous inflammations, and ends in the destruction of the nerve-medulla to a fine granular, fatty detritus. In the second form there is an interstitial exudation, and a proliferation of connective tissue, by which the neurilemma is decidedly thickened. There is rarely any formation of pus. We shall hereafter speak of the diffuse hyperæmia of the neurilemma of a wounded nerve, without any perceptible exudation, observed in some cases of tetanus.

Among the exciting causes of neuritis, the most important are injuries of the nerves, particularly punctured wounds, contusions, or lacerations. In other cases neuritis is due to the propagation of inflammation from neighboring organs. Lastly, some few cases of spontaneous or so-called rheumatic inflammation are said to have been observed.

Anatomical Appearances.—Inflammation of the neurilemma is characterized by a more or less intense redness, which sometimes depends on overfulness of the blood-vessels, sometimes on the presence of small extravasations. The neurilemma also appears relaxed, swollen, and infiltrated. If the neuritis has taken on an acute course, and led to suppuration, there is usually more or less pus in the loose connective tissue around the nerve. When the disease is chronic, the neurilemma usually appears much thickened, hard, and firmly adherent to the surrounding parts.

Inflammation of the nerve-substance is shown by its redness, swelling, and relaxation; in severe cases it is transformed to a red pulp

The neurilemma is always affected at the same time, and consequently we find the different bundles of nerve-filaments separated by the swelling of their envelopes and by interstitial exudation. Occasionally, after reabsorption of the disintegrated medulla of an inflamed nerve, only a simple cord of connective tissue is left.

Symptoms and Course.—The symptoms of neuritis cannot be clearly distinguished from those of neuralgia, or rather neuritis is one of the various causes of neuralgia. The most important symptom of neuritis is pain in the course of the inflamed nerve, extending to its peripheral terminations. The pain is increased by pressure on the nerve, and its exacerbations and remissions are usually less distinct, and the paroxysms and intervals particularly are usually less marked than in other forms of neuralgia. While this pain lasts, the sense of touch is usually lost in the parts supplied by the inflamed nerve; and this symptom may be easily explained, although it seems paradoxical on superficial examination. The morbid excitement of the trunk of the nerve, caused by the inflammation in the medulla or neurilemma, is conducted to the brain, and excites the sensation of severe pain; but the inflamed part of the nerve has become a bad conductor; hence irritation of its peripheral termination and the papillæ of touch are conducted to the brain either incompletely or not at all, and cause either no sensation or an indistinct one. At first, at the corresponding portion of the periphery, the patient has a feeling of numbness; subsequently, if resolution of the inflammation do not take place, there is complete anæsthesia to external injuries, while the pain continues. If the inflamed nerve contain motor fibres, the pain is accompanied by twitching and contraction, while the patient's power of voluntarily contracting the muscles is much affected or entirely lost. This also depends on the morbid excitability of the motor nerves being increased by the inflammation while their conducting power is lost. If the inflamed nerve lie near the surface, it may occasionally be felt as a hard cord; and we often find the skin covering it slightly reddened and œdematous. In most cases there is no fever, unless there be some other inflammation besides the neuritis.

The course of neuritis may be either acute or chronic. The more acute it is, the sooner anæsthesia and paralysis follow the neuralgia and contractions, and the more probable it is that these symptoms were caused by an acute neuritis that has destroyed the nerve. Even after resolution of the inflammation, the nerve usually remains to some extent incapable of function for a long time. When the neuritis runs a chronic course, if the nerve be destroyed, anæsthesia and paralysis occur in the same way; but, if the nerve be preserved and is only subjected to pressure from the swollen and thickened neurilemma, the

patients suffer for years from neuralgic pain or spasmodic attacks in the parts supplied by the inflamed nerve.

Treatment.—In the first place the causal indications must be fulfilled; with this view foreign bodies driven into the nerve must be removed, and inflammations in its vicinity must be carefully treated. Leeches or wet cups may also be applied along the course of the nerve; cold applications may be made, or, if the disease continue long, mercurial ointment may be rubbed in if the nerve lie near the surface. Chronic neuritis may be treated by derivatives, such as blisters, and, in obstinate cases, moxæ, and superficial linear cauterizations of the skin with hot iron. If the disease has run its course, and the nerve has not been destroyed, but its functional activity is affected, we may use electricity to restore its normal excitability, if possible, by methodical excitement of the nerve.

CHAPTER II.

NEUROMA.

Etiology.—Strictly speaking, we can only designate as neuromata those tumors originating from nerves which are chiefly, or at least to a great extent, made up of nervous elements. The tumors, not unfrequently occurring on nerves, which have developed without hyperplasia of the nerve-elements, and in which consequently the number of nerve-elements has remained the same or even actually diminished, are not neuromata, but, according to their structure, come under the head of carcinoma, fibroma, or glioma, etc. (*Virchow*). Neuromata which consist chiefly of nerve-filaments, or where these exceed the intercellular substance in amount, are termed *pure* neuromata, in contradistinction to those where the intercellular substance is in excess; this division, however, has more pathological-anatomical than clinical interest.

The etiology of neuromata is obscure. Occasionally they may be referred to a congenital or hereditary predisposition. Such cases, as well as the frequent occurrence of numerous neuromata on different nerves, and their repeated recurrence after removal, speak for the *constitutional* orgin of some neuromata. Others unmistakably result from injury of the affected nerve, by a puncture, contusion, etc. In most cases no cause can be discovered.

Anatomical Appearances.—Neuromata generally form hard, elastic tumors, from the size of a hemp-seed to that of a fist, or larger; they are usually round or oval; in the latter case their long axis lies

in the course of the nerve. *Virchow* divides them into fibro-, glio, and myxo-neuromata, according as the interstitial tissue between the nerve-filaments resembles fibrous tissue, neuroglia, or mucous tissue. They sometimes contain small cavities filled with fluid. Neuromata are occasionally seated on the nerve; again they originate from its interior, and contain more or less nerve-filaments in proportion to the distance of their origin from the centre of the nerve. The most frequent seat of neuromata is the spinal nerves; but there are examples of their occurrence on the sympathetic and cerebral nerves, particularly on the auditory. Usually there is only one neuroma, and its size is not at all in proportion to that of the nerve from which it originates. In other cases there are several neuromata on the same nerve, in still others there are great numbers, originating from the most varied nerves. Under the head of neuromata appear also to belong the painful tumors, about the size of a pea or bean, readily moved about under the skin, which are also called tubercula dolorosa, painful tubercle, or neuromantia; although their connection with a cutaneous nerve or the presence of nerve-elements in them cannot always be proved.

Symptoms and Course.—Peripheral neuromata, which alone can be recognized, show themselves as a more or less elastic tumor, seated in the course of one of the cutaneous nerves, which is only movable laterally, and is covered by the unaltered skin. Large multiple neuromata occasionally cause neither pain nor other inconvenience, so that their diagnosis can only be made by the apparent connection between the tumors and a cutaneous nerve. The case is different with the customary single small neuroma. It is often accompanied by excruciating pain extending along the course of the nerve and to its peripheral termination. This is not usually continuous, but has paroxysms with intervals of ease. Slight pressure on the tumor, often even the contact and rubbing of the clothes, increases the pain in the tumor to an unbearable extent. Severe paroxysms of pain are also induced by movement, catching cold, etc. In neuroma, as well as in neuritis, the conducting power of the nerve may be impaired, so that, besides the pain, there may be a feeling of numbness or even of more or less complete anæsthesia of the skin supplied by the nerve affected. Rarely, when the motor filaments are affected, there are twitchings and contractions and subsequently paralysis. The extension of the pain from the diseased nerve to other trunks, as well as the increased disturbance of innervation that occasionally complicates the local symptoms, is common to neuroma, neuritis, and to neuralgias of the most varied origin; we shall therefore defer their discussion to the next chapter. Neuromata generally grow slowly, and, after attaining a certain size, often remain stationary. They are among the most

painful of diseases, and may wear the patient out by the loss of sleep and restlessness induced by the pain.

Treatment.—We can never cause resolution of neuroma. The only true and trustworthy remedy is removal by the knife.

CHAPTER III.

NEURALGIA.

As we have thus far made use of the anatomical changes lying at the root of the disease as grounds of distinction, it is inconsistent to treat of neuralgia as a disease, and to class it with neuritis and neuroma. Neuralgia is a combination of symptoms, which does not depend on constant anatomical changes. Since no anatomical changes can be discovered in many cases of neuralgia, and since, in many cases, where changes have occurred, they alone are not enough to explain the neuralgia, we seem driven to the above-mentioned inconsistency, and we shall be unable to escape it in other morbid processes of the nervous system, which are not due to certain anatomical causes.

Etiology.—The sensation of pain depends on the conduction to the brain of the excitement of a sensory nerve by an abnormal irritation. Those sensations of pain, also, that are called neuralgic, depend on a propagation of this excitement to the brain. If neuralgic pains be distinguished from others, it is because they are due to the excitement of the sensory nerves by *different* irritations, or by the action of irritants at *different places*, from those causing ordinary pain. If a blow, heat, cold, or other cause acting on the termination of the nerves, induce pain, or if this be due to inflammation or other structural change of the skin, mucous membrane, or of the parenchyma of different organs, we do not call it neuralgia. But if we can discover no irritation of the peripheral termination of a nerve as the cause of the pain, or if it be probable that the irritation has affected the trunk of the nerve, the pain felt in the distribution of the nerve is called neuralgia. We may mention neuralgia of the supra-orbital branch of the trigeminus, which is not unfrequently induced by malarial infection, as the type of the form where the pain occurs in the distribution of a nerve, without the perceptible action of an irritant on the nerve itself, or on its terminations. As a type of the second form, where the pain in the parts supplied by a nerve is unmistakably due to an irritation of the nerve-trunk, we may mention the very temporary neuralgia induced by bruising the ulnar nerve near the elbow, at the part known as "the crazy-bone." It is most probable that, in those cases, also, where the injurious influence acting on the nerve escapes observation (as in neu-

ralgia caused by malarial poisoning), the action affects the trunk rather than the peripheral termination. This view is favored first by the exact limitation of the neuralgic pain to the peripheral expansions of a single nerve, and the freedom of the parts immediately adjacent, if these are supplied by other sensory nerves. This limitation would be quite inexplicable if the irritation acted on the periphery. How should we explain the constant freedom of the radial side of a finger, or the supra-orbital region of one side, from the injury that so severely affected the ulnar side of the same finger or the supra-orbital region of the other side? The view that neuralgia starts from the nerve-trunk is also supported by the fact that no idea of the variety of the irritation accompanies the pain. It is well known that the cutaneous papillæ connected with the terminations of the nerves are the chief source of the sensation of pressure and temperature. If the neuralgic pain were induced by the action of an imperceptible cause on the skin, the patients would have some impression of the quality of this irritation; they would complain of burning, piercing, or some other kind of pain. But, on the contrary, if a very cold or very hot body be applied to an exposed nerve-trunk, if we puncture or squeeze it, there is always the same sort of pain, just as in neuralgia; and, from the pain, the patient cannot tell what cause induced it. Finally, the want of benefit, in most cases, from division of nerves, indicates that the seat of the disease is to be sought for in the trunk or branches, not in the peripheral expansions of nerves. We do not know what physical or chemical changes of the nerves cause their morbid excitement in neuralgia. We may even suspect that they do not consist in any very evident deviations from the normal, for these remove the excitability, but that injuries only act as causes of neuralgia, when they exercise a comparatively slight irritation on the nerves. If we find a nerve, which was affected with neuralgia, much changed at some point, we may be sure that this was not the starting-point of the pain, but that it originated from some point higher up, where no changes of structure can be made out with the naked eye or with the microscope.

The predisposition to neuralgia varies with the person. A morbid increase of excitability of the entire nervous system, so-called nervous debility, of which we shall speak hereafter, and which is seen more frequently in women than in men, in bloodless and debilitated persons than in the full-blooded and strong, appears to lead to the occurrence of neuralgia in some persons more readily than in others.

The exciting causes—that is, the irritations which, by their action on the nerve-trunk, induce the neuralgia—are partly known, partly unknown. We are not justified in distinguishing cases from unknown causes as "genuine" or "pure" neuralgia. The pain caused in the

finger by striking the ulnar nerve near the inner condyle of the humerus, or in the toes by a blow on the sciatic nerve where it escapes from the sciatic notch, is also a genuine, pure neuralgia. Practically it would be better to designate as pure neuralgia those cases that continue after the cause that induced them has ceased to act. Among the injuries that may be regarded as direct causes of neuralgia, the most frequent are: 1. Wounds of the nerves from sharp instruments, such as lancets, needles, etc.; a complete solution of continuity is far less dangerous than these punctured wounds. 2. Irritation from foreign bodies that have entered and become incapsulated near the nerve; obstinate neuralgia has very often been observed as a result of the irritation caused by pieces of musket-balls. 3. Compression of the nerves by contracted cicatrices. 4. Pressure on the nerves by aneurisms, exostoses from bones and teeth, and tumors, particularly carcinoma. 5. Neuroma, described in the last chapter. 6. Overfilling of the veins in the vicinity of the nerves where they pass through bony canals. From the predisposition of the left side of the body to intercostal neuralgia, *Henle* concludes that the latter cause has a material influence on the occurrence of neuralgia. The left side differs from the right in the arrangement of the venous circulation, so that the blood must make a circuit (from the hemiazyos vein into the azyos) to pass from the veins of the spinal cord into the vena cava; if there be any obstacle to the escape of blood from the heart, it must necessarily have a worse effect on the left side than on the right. The theory that neuralgia often depends on dilatation of the venous plexuses surrounding a nerve where it passes through an opening in the bone, is also supported by the fact that the first branch of the trigeminus (whose relation to the venous plexuses in its vicinity resembles that of the intercostal nerves) is far more frequently the seat of neuralgia than the second or third branch, where this is not the case. In the so-called rheumatic neuralgias, caused by catching cold, we can find no material changes in the neurilemma to explain the irritation of the nerve; nevertheless, although hypothetical, it is very probable that this rheumatic neuralgia is due to a hyperæmia and œdematous swelling of the neurilemma, which disappears after death. Lastly, we may mention, as causes of neuralgia, poisoning by metallic substances, such as mercury, lead, copper, etc., as well as by malarial infection. It is perfectly inexplicable, in these cases, why the irritation from a constitutional disease should only affect a very circumscribed nerve-tract.

Symptoms and Course.—In neuralgia we may distinguish two forms of pain: one continuous, increased by pressure, confined to circumscribed points in the course of the nerve (points douloureux, *Valleix's*), not very severe but annoying pain; the second occurs in paro-

ysms, spreading from a point along the course of the nerve; the pain is terrible and almost unbearable. The points douloureux occur particularly where the nerve escapes from a bony canal, or from fascia that it has perforated, and approaches the surface. These spots seem larger to the patient than they prove to be when we mark them out by pressing the finger around. *Budge* made some very interesting observations on the excitability of the motor nerves of frogs at different places; he found some spots very excitable while the parts immediately next to them were very slightly so; perhaps *Valleix's* points douloureux on the sensory nerves correspond to those that *Budge* found peculiarly excitable in the motor nerves. While *Valleix* almost always found the points douloureux in neuralgia, other observers have just as constantly failed to find them. The paroxysmal pains sometimes pass downward, sometimes pass upward along the course of the nerve, so that there has been a division made into neuralgia descendens and ascendens, the latter being far the more rare. Patients usually say that the pain is not superficial but deep. It is rare for the paroxysms of pain to be limited to one small twig of a nerve; usually several twigs of one branch, but only rarely all the twigs of one nerve participate in the affection. It is very remarkable that not unfrequently the neuralgia extends from one nerve to another that has a different origin. From the laws of conduction we should suppose that such a transfer could only occur in the central organs, through the ganglia; but the observation that neuralgia not unfrequently extends from a cerebral nerve, as the trigeminus, to a spinal nerve, as the occipital, renders the transfer in that way very improbable; and we must content ourselves with having mentioned the curious fact.

Anomalies in the distribution of blood, in the secretion and in the nutrition of the parts supplied by the affected nerve, are not unfrequently observed without our understanding how the morbid excitement of the sensory can cause abnormal excitement of the vasomotor nerves. At the commencement of neuralgic attacks we occasionally see the skin become pale, more frequently at the height of the attack that it reddens, that the secretion from the nasal mucous membrane, conjunctiva, and from the lachrymal and salivary glands, is increased In the same category come the exanthemata that develop in the course of the affected nerve in some neuralgias, particularly in intercostal neuralgia (herpes zoster), and lastly, the atrophy or excessive development of fat in parts supplied by the affected nerve, when the disease has lasted a long while. The morbid excitement of the sensory nerve is rarely transferred through a gland to a motor nerve. We must beware of carelessly considering the twitchings of patients, during their attacks of pain, as reflex symptoms.

Except in the cases resulting from malaria, the course of the disease is chronic. It is rarely regular, the individual attacks are marked by paroxysms of pain interrupted by free intervals, and in the general course of the disease we are apt to see remissions and exacerbations. At times the attacks of pain are more frequently repeated and are more severe, at others, they return less frequently and are less severe. It is only in the neuralgia caused by malaria, in the so-called febris intermittens larvata, that the paroxysms of pain show a regular type. In other cases the type is irregular, and the attacks not only recur spontaneously, that is without assignable reason, but they are excited by various recognizable causes. Among these are irritation of the skin which the affected nerve supplies, by pressure, friction, cold, heat, etc. Slightly touching the skin often appears to induce attacks of pain more readily than heavy pressure does. Movements of the parts where the pain is located, i. e., chewing in neuralgia of the trigeminus, walking in neuralgia of the sciatic, coughing and sneezing in that of the intercostals, excite attacks of pain. Mental excitement occasionally has the same effect. I treated one old gentleman, with neuralgia of the trigeminus, who had such a severe attack of pain every time I entered his room, that he could not salute me for some time. Each attack of pain usually lasts only a few seconds. But these short attacks are often repeated several times in the course of one or a few minutes, and then cease for a while, so that in fact we may say that in neuralgia long attacks occur which are composed of a number of short paroxysms. As we are almost compelled to believe that the irritation acting on a nerve, which causes the neuralgia, acts continuously, the intervals between the pains appear enigmatical: for their explanation we must refer to the physiological fact that the severe irritation of a nerve exhausts its excitability for a time; then in neuralgia, states of great excitement would alternate with states of diminished excitability. This hypothesis is to some extent supported by the observation that, after severe attacks of pain, the peripheral terminations of the nerve are for a time less sensitive to irritation, as if their excitability were diminished, as well as by the observation that, after inducing a severe neuralgic attack by continued pressure on a painful point, a repetition of the pressure does not induce a second attack. The gentleman above mentioned also had attacks of pain as soon as he began to eat. In order that he might be able to eat at meals, he began them by biting vigorously on some hard bread; this induced a severe attack, but after it passed over he remained free from pain during the remainder of the meal.

Neuralgia may continue for many years. Except in the cases caused by malaria or the so-called rheumatic neuralgia, complete cure

is by no means frequent. Even the termination in permanent anæsthesia is comparatively rare, although we should *a priori* regard it as the most frequent, since it would seem very probable that continued irritation must finally destroy the nerve. In many cases neuralgia, particularly certain forms of it, remains stationary and lasts till death. This is not apt to result from the neuralgia itself, but from accidental complications, or from the disease causing the neuralgia.

Treatment.—Where the neuralgia is induced by pressure on, or compression of, the nerves by foreign bodies, tumors, or contracted cicatrices, the causal indications require surgical interference. The circumstance that neuralgia occasionally continues after the removal of foreign bodies and tumors from the vicinity of nerves, should not deter us from operation; since we cannot know beforehand whether the neuralgia has become "habitual," that is, whether the injurious influences that have acted on the nerve have affected it permanently, and have induced a state that will not subside after removal of the cause. The so-called antirheumatica are of little use in rheumatic neuralgias. These would be more successfully treated by the methodical use of artificial or natural warm-baths. Numbers of patients with rheumatic neuralgia seek relief, and some find it, at Wildbad, Baden-Baden, Wiesbaden, and other warm springs. Where the disease is due to malaria and has a regular type quinine, the antidote to malarial poisoning has a very brilliant effect. Sulphur-baths and the internal administration of sulphur have a peculiar reputation in the neuralgias caused by poisoning with copper, mercury, and lead. Finally, the causal indications require the treatment of the disposition for neuralgia; and, as we know, to some extent at least, what it depends on, we cannot unfrequently fulfil this indication successfully. We cannot regard carbonate of iron as a specific for neuralgia, but, when poverty of the blood is one of its chief causes (or, as *Rademacher* has it, "when the neuralgia is an iron affection of the constitution"), the carbonate and other preparations of iron often have a surprising effect. In the same way neuralgia may often be benefited by modes of treatment that greatly modify the change of tissue and the nutrition. Where we cannot remove the causes of neuralgia, the indications from the disease require that we should attempt to equalize the disturbances of nutrition on which it depends, or to destroy the excitability of the nerves, or finally to prevent the propagation of the morbid excitement to the brain.

Among the most effective modes of treatment for this purpose is the use of electricity. Excellent results are not unfrequently attained both by the induced and constant current. Dr. *Leube*, in his inaugural dissertation in 1862, reported a number of cases of obstinate neu-

ralgia that were cured at my clinic by the induced current. Since then, my experience in the result of this treatment has greatly increased, and I may combine the results in the following propositions: 1. In treating neuralgia with the induced current, it is best to employ the metallic electrodes known as the electric brush: while one electrode containing a moistened sponge is held in one of the patient's hands or against any part of his body, we stroke the brush along the course of the affected nerve; if there are any points douloureux we allow the brush to remain over them rather longer (electric moxæ). 2. Many cases of neuralgia, which had been previously treated without benefit by the most varied remedies, were completely and permanently cured in from twelve to twenty applications, or even sooner. In other cases no benefit or cure was effected. 3. The first sitting shows whether the neuralgia can be cured by the induced current. We can only expect a cure where the pain is decidedly relieved or entirely disappears immediately after the first electrization, even if it should only be for a short time; if this temporary result do not take place, the continuation of the treatment will also prove ineffectual. The application of the induced current as above directed is very painful; and it is only after the patient has actually experienced benefit that he suffers it with patience, and even then he moans and whimpers during the application. An erythema, that lasts for some time, forms where the current is applied. I shall not attempt to decide whether the induced current acts by derivation to the skin like the linear cauterizations advised by *Valleix*, or as blisters and irritating frictions, or whether it acts in some other way.

The constant current is far more effective than the induced in neuralgia. Some cases that have been treated without result by the induced current have been cured by the constant, while I have never seen the reverse. I place both poles along the affected nerve, and, without attending to the course of the current, hold the zinc pole on the most painful part, and on those parts where the nerve approaches nearest to the surface, as at the supra or infra-orbital foramen, or at the zygomatico facial foramen, or at the sciatic notch. If it be possible to get the nerve between the poles, as in the cheek or nose, I introduce one pole into the mouth or nose to the point whence the pains radiate, while I place the other at the corresponding point on the skin. At first, the application of the constant current is not particularly painful, but an unpleasant, burning, piercing pain soon commences and gradually increases; where the number of elements is large, it may become unbearable. The changes induced in the skin at the point of application of the constant current, if the electrodes be applied for a length of time, are far greater than those caused by the induced current

They not only consist in a lively erythema, a decided swelling of the skin, and an increase of the subjacent tissue, but papules and blebs arise on the skin, particularly at the positive pole; if the action continues long, the surface of the elevations sloughs off. These changes take place not only at the point of application, but occur equally or at least similarly in the deeper parts, as is shown not only by the increase in volume of the subcutaneous tissue and muscles, but also by the following experiments (*Erb*), which show a great deal about the mode of action of the constant current in neuralgia and other neuroses, as well as in some diseases of the muscles, joints, etc. If we cross the forearms, placing the volar surfaces in contact, and apply the electrodes to their dorsal surfaces, there will be reddening not only of the parts to which the electrodes are applied, but of the corresponding points on the anterior surfaces. Generally, relief immediately follows the application of the constant current just as it does that of the induced current; but occasionally the pain is at first increased, and that should not always induce us to stop the treatment. There seems to me no doubt that the curative action of the constant current, in most cases of neuralgia, is to be explained by the modification of the circulation, endosmosis or change of tissue in the diseased nerve, its neurilemma, or the parts around; this "catalytic action" may result from the chemical disintegration induced through the nerves, or the attraction of the constituents of the nutrient fluids toward the pole, or it may occur in some other way. The swelling of the skin, and the eruption of nodules and blebs on it after the application of the constant current, do not prove its action on the vasomotor nerves any more than the redness of the skin after a mustard plaster shows a similar action in oil of mustard. I consider it perfectly unjustifiable, in most of the recent writers on electrotherapeutics, to make a distinction between the effect of the constant current in neuralgia, paralysis, etc., and its effect in disturbances of nutrition. In most cases of neuralgia or other nervous diseases, just as in the affections of the muscles, joints, etc., where galvanism has proved useful, there is no molecular change or any alteration in the electrical state of the nerves, but, as *Remak* distinctly asserts, there are disturbances of nutrition, anomalies of circulation and structure, exudations, etc. For the sake of the good cause, I cannot help regretting the numerous attempts that have been made to explain the benefits from the constant current in disease of the nerves and muscles, by referring to the laws concerning the contractions at the opening and closing of the current, concerning anelectrotonus and catalectrotonus, or concerning the results of irritation and division of the sympathetic, instead of resting solely on the evident results of clinical observation. These very imperfect doctrines, which are founded

on the results of experiments made on healthy frogs and rabbits, fur nish no useful data for the explanation of cures induced in diseased human beings by the employment of electricity. Supposing, in a paralysis, neuralgia, or anæsthesia, we can succeed in changing the electrotonus of the affected nerve, which I do not at all deny, there is not the slightest probability that we shall thus remove the textural changes which lie at the root of the existing paralysis, anæsthesia, or neuralgia. On the contrary, if cure results, we may make up our minds that, besides the changes of the electrotonus, some other action has been induced. And supposing we succeed, by a few minutes' irritation of the sympathetic, in contracting the vessels supplied with nerves from that part, for the length of the sitting, which I also shall not deny, it is just as improbable that we should thus remove a disturbance of innervation existing in the parts supplied by the contracted vessels. If application of the constant current to the sympathetic causes a cure, we may suppose that the disease depended on some disturbance of nutrition of the sympathetic, that has been removed by the catalytic action of the current. The marked difference between the constant and induced current in regard to their chemical action on water, solutions of salt, albumen, etc., has long been known, and I am fully convinced that the introduction of the former into practice is one of the most valuable advances of modern times, and that *in the constant current we have a means, more powerful than any other, of modifying the nutritive conditions of parts that are deeply situated.* But I fear that the rationalistic and doctrinal teachings about galvanotherapeutics, which are recently so popular, and the attempts to make this so "exact," may interfere with moderate and experimental observation, and injure the popularity of an important remedy.

In the same class as electrical treatment come the blisters, moxæ, actual cautery, and cutaneous irritants, which are used as derivatives to the skin, and which are being more and more supplanted by electricity. Superficial linear cauterization is considered, particularly in France, as one of the most effective of remedies.

Among the means by which the excitability of the nerves is destroyed, we shall first mention cold. Besides compresses of cold water and ice, lotions with ether and liquor Hollandicus are used; these induce cold by their rapid evaporation. If they are more effective than ice-compresses, it is because they are breathed in at the same time, and to some extent stupefy the patient. Cold is a valuable palliative; we cannot generally continue its use long enough for a radical cure. The *narcotics*, particularly in the form of hypodermic injections of solutions of morphia, are at present the most common remedies for neuralgia. Formerly, if the local action of morphia was desired, a

blister was applied, the resulting vesicle was opened, and the desired dose of morphine was sprinkled on the denuded spot, or else the morphine was mixed with saliva and inoculated into the skin. The introduction of hypodermic injections, instead of these troublesome procedures, was a great event, and was regarded as an immense advance in treatment. It was thought that the affected nerves might now be narcotized with facility. The results were perfectly surprising. In numerous cases, instead of writing a prescription of doubtful efficacy, the physician could free the patient from his pain in a few minutes. Moreover, it soon became evident that, not only in neuralgia, but in many other painful diseases also, this effect followed the hypodermic injections of morphine; and, secondly, that it did not make much difference whether the injection was made at the seat of the pain or at some other point. I know many physicians who never go out to their practice without a *Pravaz's* syringe and a solution of morphine in their pocket, and who usually bring the morphine-bottle home empty. It cannot be denied that the hypodermic injection of solution of morphia is sometimes abused. From this abuse we have become acquainted with a form of chronic morphine-poisoning that was previously little attended to. If injections of morphia have been made for some time, and the dose has been increased more and more, independent of the return of the pain, the patients begin to feel an absolute need of the injections. They feel dull, and complain of an undefinable weakness, discomfort, trembling, etc. Some describe their state as resembling that after a debauch. Indeed, the condition before and after the injection often reminds us most strikingly of that of a toper, before and after his first glass of spirits in the morning. But these bad results may be avoided by the careful use of hypodermic injections of morphia. It is doubtful whether they have any local action; but it is certain that the general effect of the morphine is much more complete and precise, if it be injected under the skin, than if it be administered internally, and we must regard the hypodermic injection of $\frac{1}{6}$ to $\frac{1}{3}$ of a grain of morphine as an invaluable palliative for neuralgia. Next to the employment of cold and narcotics, come frictions of the skin with veratrine ointment (gr. iv—x to fat ʒ j), or aconite ointment (gr. j to fat ʒ j). After using veratrine ointment, the patients feel a peculiar prickling in the skin, which occasionally benumbs the pain. [A mixture of equal parts of chloral hydrate and camphor forms a clear liquid, which painted on the part relieves pain.]

We have stated that a third requirement of the indications from the disease is to prevent the conduction of excitement from the irritated nerves to the brain. The most effective mode of doing this is by dividing the nerve between the brain and the affected part, or by

cutting out part of the nerve, since it readily heals up if simply divided. The want of success in this operation is chiefly due to a division of the wrong nerve, or to its being divided at the wrong place, that is, to the distal side of the point whence the pain orginates (*Bruns*). Unfortunately, the division at "the right spot" is in most cases entirely impossible, because the pain originates from a point above which the nerve cannot be reached. Cauterization of the nerve, for the purpose of disconnecting it from the brain, should be given up; compression is only to be used as a palliative. The least benefit is obtained from the specifics recommended in neuralgia, such as arsenic in the shape of *Fowler's* solution, the preparations of zinc, particularly the valerianate and hydrocyanide, nitrate of silver, and other metallic and vegetable nervines. There are cases recorded where each of these remedies is said to have produced brilliant results, but these examples are few, and are far outweighed in number by those where they have had no effect. Up to the present time it is absolutely impossible to determine exactly in what cases these remedies are useful.

CHAPTER IV.

NEURALGIA OF THE TRIGEMINUS—PROSOPALGIA—TIC DOULOUREUX—FOTHERGILL'S FACEACHE.

Etiology.—Next to the sciatic, no nerve is so often the seat of pain as the trigeminus. This is partly because many branches of the trigeminus pass through narrow canals and openings, where they may readily be compressed, partly from the distribution of the nerves to portions of the skin where they are more exposed to cold than other nerves are. *Hyrtl* is doubtless correct in supposing that the passage of the branches of the trigeminus through narrow openings of the bones has much to do with the occurrence of facial neuralgia; for see the great immunity to the disease of those branches which pass through the wide sphenopalatine foramen to the nose, compared with its frequent occurrence in the infraorbital, zygomaticus malæ, and superior and inferior dental branches.

Only in a few cases have foreign bodies under the skin (in one celebrated case of *Jeffreys* there was a piece of porcelain) and tumors pressing on the branches of the trigeminus been found as causes of prosopalgia. Somewhat more frequently changes in the bony canals can be found to explain the morbid excitement of the nerves traversing them; among these are exfoliations of a bony wall in neuralgia of the infraorbital, exostosis of the root of a tooth in neuralgia of the inframaxillary, general thickening of the bones of the skull with con-

traction of the foramina through which the nerves pass, or even inflammation and projections on the bones. Finally, in some cases, aneurisms, tumors, thickening of the dura mater, exostoses within the skull, which pressed on the trigeminus, have been found as evident cause for obstinate and extensive neuralgia of the branches of that nerve. I do not know a single case that shows an unequivocally central origin of facial neuralgia. Among the cases described by *Romberg*, in one case of neuralgia of the trigeminus that had lasted for twenty-six years, it is true there was a small diseased spot in the pons, but there was at the same time an aneurism of the carotid compressing the trigeminus, which would fully have accounted for the neuralgia.

Far more frequently no material causes can be found for facial neuralgia. In such cases it is very probable, but cannot be proved, that the disease is occasionally caused by catching cold, which induces hyperæmia and slight œdema of the neurilemma that disappear after death. Cases of hæmorrhoidal facial neuralgia and those from suppression of perspiration and exanthemata are very problematical; arthritic cases are somewhat less so. Neuralgia of the trifacial is the most frequent form when the disease is due to malaria.

The rather worthless results of statistics show that facial neuralgia is rare in childhood, most frequent between the thirtieth and fiftieth years, and somewhat more common in women than in men.

Symptoms and Course.—Of course, the pain from which patients with neuralgia of the trigeminus suffer is more extensive in proportion as the branch of the nerve affected is larger; and conversely, from the limitation of the pain to a circumscribed spot, we may decide that a very small branch is the seat of the disease. Moreover, as the filaments of a nerve are the more numerous the nearer we go to its origin in the brain, and become fewer as it approaches the surface, it follows that, when the pain is very limited, we may consider its origin as peripheral; where it is very extensive, we may decide that the injurious influence is in the skull itself. In fact, in neuralgias caused by pressure on the trunk of the fifth pair, pain has been observed in all parts supplied by its sensory filaments, in the anterior surface of the ear, in the skin of the forehead, temple, face, in the orbit, nose, palate, body of the tongue, floor of the mouth, teeth, and probably in the dura mater. *Valleix* mentions numerous points douloureux in facial neuralgia; we shall only call attention to three of them, which lie nearly in a vertical straight line, and correspond to the supraorbital foramen, the anterior opening of the suborbital canal, and the mental foramen. If the neuralgia be seated in the *first* branch of the trigeminus, the pain spreads particularly in the branches of the supraorbital, and affects the forehead, eyebrows, and upper eyelid. In some rare cases the pain is chiefly in

the eye, and, from participation of the infratrochlearis, at the inner canthus and caruncula lachrymalis. The twigs of the first branch, which go to the lachrymal gland and conjunctiva, explain the increased secretion of tears and redness of the conjunctiva, almost always observed in neuralgia of that branch, particularly on remission of the paroxysms. If the *second* branch of the trigeminus be the seat of the neuralgia, the pain is usually most severe in the parts supplied by the infraorbital, that is, in the lower eyelid, alæ nasi, upper lip, and teeth of the upper jaw. The attacks of pain in these cases are sometimes accompanied by watery or mucous secretion from the nasal mucous membrane. Neuralgia is rare in the course of the *third* branch of the nerve: this is particularly true in the course of the auriculo-temporal and lingual branches; it is somewhat more frequent in the inferior alveolar, especially in the mental, after it escapes from the foramen; then the patient has pain in the chin and lower lip. Salivation often accompanies neuralgia of the third branch; this symptom agrees perfectly with the experiments of *Ludwig* (see vol. i.). Occasionally the neuralgia chiefly affects those twigs of the second and third branches that accompany the ramifications of the facial nerve. This explains why the facial nerve itself was formerly often considered as the seat of the neuralgia.

In facial, as in other neuralgias, the patients suffer partly from a permanent dull pain located at different points of the trigeminus; partly from attacks of agonizing pain, which occur suddenly, then cease as suddenly, in half a minute or so, and again return till the attack, composed of these short twinges, "Tics," is over. The face often twitches during these attacks, but does not usually do so involuntarily. Parson *Barth*, who has given a very careful account of his own facial neuralgia, was even able to continue preaching during the attack. The attacks sometimes occur spontaneously, and, except in the cases due to malaria, they are irregular; sometimes they are induced by the causes previously mentioned, such as speaking, sneezing, gaping, blowing the nose, by using too cold or too warm food, occasionally even by any attempt to chew. During the attack the face is usually reddened, its temperature increased, and there is active pulsation of the arteries. To the cases observed by *Brodin* and *Romberg*, where, after long duration of severe facial neuralgia, the countenance of the patient was greatly disfigured by swelling and development of fat, I may add the case of one of my Magdeburg colleagues, in whom there was decided disfigurement, particularly by swelling of the lower lip, from facial neuralgia of one year's duration. In old cases of the disease other disfigurements of the face have also been observed, such as increased thickness and prickliness of the beard, acne pustules, etc.

The duration of the disease is rarely short, except in the regular intermittent malarial affection; in almost all other forms it usually lasts a long while, even half a lifetime, or more. Among the terminations, besides recovery, severe melancholy occurs, which may drive the patient to suicide. Death also occasionally results from the cause of the disease, but not from the disease itself.

Treatment.—For the treatment of facial neuralgia we have little to add to the rules given for the treatment of neuralgia in general. Only in a few cases can we cure prosopalgia quickly, by the removal of foreign bodies, tumors, and contracting cicatrices. Extraction of teeth does not often prove of benefit. Usually the unfortunate patient has one tooth after the other drawn, without the slightest relief to the pain.

In recent cases, caused by catching cold, *Valleix* urgently recommends flying blisters and superficial cauterizations with the hot iron. If the disease be due to malaria, large doses of quinine are almost always of service. The administration of Fowler's solution (four to six drops every three hours) is only indicated when quinine has failed. In decided anæmia, we may give preparations of iron, and, when we conclude that there is some other constitutional disease, whose nature we cannot determine, we may order alterant mineral waters and baths.

Concerning the employment of electricity, cold, veratrine, and aconite, as well as of morphine, particularly in the form of subcutaneous injection, the same is true in facial as in other forms of neuralgia. I have seen two cases of tic douloureux cured by the constant current; one of them was of thirty years' standing, and in the other eleven operations, some of them severe ones, such as the ligation of the carotid, resection of the superior maxilla, etc., had been performed without benefit. In no form of neuralgia is division of the nerve, or excision of part of it, more frequently done. After this operation had for a time almost gone out of use, *Bruns* has shown, by a careful examination of the cases where it had been done, that, after excluding the cases where its want of benefit depended on error of diagnosis or operation, and after excluding the cases where the recurrence of the pain was to be regarded as a new attack, not as a relapse, there was a considerable number of cases where the neurotomy caused either a complete and permanent benefit, or at least a temporary one, for a few months or years. According to *Bruns*, the operation (whose performance we will not describe) is indicated when the pain is very limited, and it is probable that its exciting cause is situated at a point beyond which the nerve is accessible to the knife, when other treatment has proved fruitless, and when the pain renders the patient unfit for business. There is also an indication to operate when we cannot

hope to divide the nerve between the point of disease and the brain, but when the pain never occurs spontaneously, being always the result of some external irritation acting on the peripheral terminations of the nerve. In such cases the operation may, to some extent, protect the patients from the exciting causes of their attacks of pain. The temporary compression of the affected nerve and the artery supplying it has also sometimes proved an excellent palliative, and deserves a trial in suitable cases. Among the so-called specifics, *Romberg* speaks most highly of arsenic; under its use he saw the most evident and speedy benefit in those cases that occurred in hysterical women, or from disease of the sexual organs. Its effect was the more certain, the more anæmic the patient. *Romberg* also saw temporary benefit from nitrate of silver in large doses (gr. j). *Bell*, who, as *Watson* says, "shot an arrow at random," claims to have had excellent results from the internal administration of croton-oil with compound extract of colocynth. Among the narcotics, besides the preparations of opium and morphine, belladonna, stramonium, conium, and their alkaloids, particularly *Meglin's* pills (consisting of equal parts of extract of hyoscyamus and flowers of zinc), have a good reputation; we begin with a two-grain pill, morning and evening, and increase to twenty or thirty of them daily.[1]

CHAPTER V.

HEMICRANIA—MIGRAINE.

The combination of symptoms designated as migraine is difficult to explain. It is even doubtful if we are justified in classing it among the neuralgias, as is almost universally done. The almost constant occurrence on one side, of the headache (to which hemicrania owes its name), the paroxysms and free intervals observed in the disease, the negative results of anatomical examination, are the chief reasons for this view. On more carefully examining into the general course of the disease, and the course of individual cases, however, this view is not supported. There is no variety of neuralgia which, beginning, like migraine, in childhood, and lasting into advanced age, only attacks the patient a few times a year during this period, and, in these attacks, shows a steady increase and diminution of the pain, but never an instantaneous occurrence or disappearance. Still other objections to the consideration of migraine as neuralgia will appear from the description of the symptoms and course. Whether migraine be a neuralgia or not, the pain must result from excitement of sensory filaments, but it is doubtful whether this excitement occurs in the filaments from the

trigeminus to the dura mater, in the sympathetic filaments accompanying the vessels, or in the brain itself. According to a fanciful hypothesis of *Du Bois-Reymond*, which Dr. *Möllendorf* has carried still further, the attack of migraine depends on a dilatation of the branches of the internal carotid, the result of abnormal innervation of the vessels from the ganglion supremum of the sympathetic. According to this view, the brain would, during an attack, be in the same condition as the ear of a rabbit that has had the cervical portion of the sympathetic divided.

Migraine is a very common disease, so that, in a moderately-extensive practice, we may usually observe a series of cases for years, and be again and again reminded by them of the impotence of our art. The disease occurs in both sexes, but is far more frequent among women than men. Like gout, migraine is considered a fashionable disease, but it also occurs among the lower classes, and is for them a peculiarly-distressing disease, as they cannot lie by for it. In most cases its commencement dates from the time of going to school, if not from early childhood. Probably in half of the women affected with migraine, the attacks occur at the menstrual period, or immediately before it. In other cases the attacks are unmistakably due to mental excitement. I treated one lady, who never had a party without suffering from migraine next day; another was affected in the same way after each visit to the theatre. In some cases a severe attack, particularly if it end with sick stomach, results in a certain immunity to relapses. The first lady above mentioned had her guests invited the day she had migraine, so that she might be certain of being able to receive them the following day. In the cases that I have seen, the attack could rarely be referred to an error of diet; but, on the other hand, the pain and general disturbance were almost always increased if the patient took any food, no matter how digestible, during the attack.

After feeling perfectly well the day before an attack of migraine, the patients perceive the first symptoms as soon as they awake, or immediately afterward. They feel heavy and depressed, are uneasy and irritated, complain of slight chilliness, are inclined to gape, usually have no appetite, and have a slimy taste in the mouth. Besides the above, there is headache, which is almost always limited to one side, increases rapidly, and becomes almost unbearable. The relaxation and pain drive the patients to bed; they are very sensitive to light and noise; hence they seek the darkest and most retired chambers. They dislike all visits, even that of the physician, during the attack The pulse is usually slower. In many cases, at the height of the attack (in some patients at every attack), there is nausea, and, after great

retching, vomiting, by which quantities of a very bitter greenish fluid are evacuated. Patients who have frequent attacks of migraine, usually long for the commencement of vomiting, and even try to excite it by irritation of the pharynx. Generally, toward evening, rarely earlier, the patient falls asleep, and awakes next morning free from pain, although much depressed. The disease never threatens life; but, although the attacks sometimes occur at shorter or longer intervals, patients rarely entirely recover from the disease. In women alone, particularly those who have the migraine at their menstrual periods, the disease occasionally ceases at the change of life.

Watson asserts that "four to six drops of liquor arsenicalis, given three or four times daily, with attention to the digestion, effected a cure in ten cases of hemicrania;" but this assertion is a solitary one; most other observers say that the disease generally resists all treatment. Nor have I seen any decided benefit from arsenic, pulsatilla, marsh-trefoil, or from the very expensive citrate of caffein (which is also called a specific), of which we prescribe pills (℞ caffein citr. gr. x, ext. grani [*Triticum repens*] ʒj; ft. pil. 10), and, to cut short the attack, give one or two of these pills every hour on the first symptoms, or from the paulinia sorbilis, which is prescribed in the form of pasta guarana ʒss—ʒj. One patient prepared for herself an infusion of unroasted coffee, and, as long as she drank this daily, appeared to have the attacks more rarely and less severely. Another patient escaped the attacks as long as she took sea-baths at Häringsdorf; but, when she returned home, they began again. In most cases we can do nothing but attend to existing disturbances of the general health and of the digestion, and the chances of benefit from treatment are much greater where we can discover any such disturbances. During the attack we should spare the patients from the use of any remedies, and let them take nothing but water. It is best for the patient not to trv to defy the attacks, but to go to bed early.

CHAPTER VI.

CERVICO-OCCIPITAL NEURALGIA.

Cervico-occipital neuralgia, or neuralgia of the sensory nerves of the occiput, neck, and nape of the neck, which originates from the first four cervical nerves, is far more rare than facial neuralgia. The cases known, up to the present time, are not sufficiently numerous to give us any thing definite concerning the etiology. From the observations of *Valleix*, according to which the disease often began after prolonged exposure to cold, and from one case where relapses often occurred, and

came only in winter, as well as from the generally favorable course and good result of treatment, it seems probable that this disease most frequently results from catching cold. Diseases of the vertebræ also appear to induce this form of neuralgia, in some few cases, by pressing on the veins as they pass out of the vertebral canal; and swollen lymphatic glands, deep in the neck, may cause it by pressing on the cervical plexus and the occipitalis major.

In cervico-occipital neuralgia, patients also complain of a continued dull pain, limited to small spots, which is, from time to time, accompanied by attacks of very severe lancinating pain shooting out in various directions. According to *Valleix*, these isolated painful spots are: 1. An occipital point located below the occipital bone, between the mastoid process and the first cervical vertebra, corresponding to the point where the nervus occipitalis major [posterior occipital] perforates the complexus muscle and becomes subcutaneous. 2. A superficial cervical point, somewhat above the middle of the neck, between the trapezius and sterno-cleido-mastoid muscles, corresponding to the point of exit of the chief cervical nerves. 3. A mastoid point, lying behind the mastoid process, corresponding to the occipitalis minor and auricularis major nerves. 4. A parietal point near the parietal protuberance. 5. An auricular point in the auricle. In the attacks the pain darts from these points toward the occiput, the posterior, and upper part of the neck, anteriorly to the face, and, occasionally, downward, toward the shoulder. Cervico-occipital neuralgia is not unfrequently complicated with prosopalgia and neuralgia of the brachial plexus. The attacks sometimes occur at irregular intervals, sometimes from movements of the head, or other slight causes. They are rarely so severe as the attacks of tic douloureux. Disturbances of nutrition in the territory of the cervical nerves are exceedingly rare, if they ever occur. Cervico-occipital neuralgia is far less obstinate, and is rarely so permanent as facial neuralgia.

In this affection division of the nerves has not been tried. In recent cases blisters are recommended, particularly by *Valleix*, as well as *Meglin's* pills and all other remedies that are used in prosopalgia.

CHAPTER VII.

CERVICO-BRACHIAL NEURALGIA.

By cervico-brachial neuralgia we mean a neuralgia located in the sensory twigs of the brachial plexus, which is composed of the lower four cervical and the first dorsal nerves.

This form of neuralgia has perceptible causes far more frequently

than other forms. Among these are injuries of the peripheral branches of the brachial plexus in the arm or hand from lancets and other pointed instruments, pressure on the nerves from fragments of bullets, contusions, neuromata, etc. The brachial plexus itself is occasionally compressed in the axilla by swollen lymphatic glands, under the clavicle by the callus of a fractured rib, by aneurisms of the subclavian or arch of the aorta. Finally, the nerves forming the brachial plexus may be pressed upon and irritated just at their escape from the spinal canal, by diseases of the vertebræ. In other cases the irritation acting on the brachial plexus or its branches cannot be perceived, and we have to refer the neuralgia to a rheumatic affection of the neurilemma, or to an imperceptible alteration in the nerves, from over-use of the muscles in knitting, playing the piano, etc. Lastly, we must mention that attacks of angina pectoris are usually complicated with pain in the course of the brachial plexus. The transfer of the morbid excitement from the nerves of the heart to those of the arm is best explained, according to my idea, by the intervention of the nervus cardiacus magnus and parvus, as they originate from the middle and inferior cervical ganglia which are connected by many twigs with the inferior cervical nerves.

In some cases the pains spread over a large part of the sensory filaments of the brachial plexus, in other cases they are limited to the axillary region and the upper part of the arm; occasionally they follow exactly the distribution of the ulnar, radial, or musculo-cutaneous nerves. *Valleix* most frequently found a point douloureux in the axilla, and one for the ulnar nerve, the point between the inner condyle and the olecranon, and another near the ulna above the hand where the ulnaris becomes superficial; and for the radial nerve the point in the arm where this nerve winds around the humerus, and a second one at the lower end of the radius just above the hand. The lancinating pains, particularly those extending to the fingers supplied by the affected nerve, are severe and recur very frequently, so that the intervals of perfect freedom from pain are usually shorter than in other neuralgias. The pain is generally accompanied by a sense of formication and numbness in the fingers, which lasts longer than the attack. Disturbances of nutrition in the parts supplied by the morbidly-excited nerve occur in some cases of brachial neuralgia in the shape of exanthemata (pemphigus, urticaria), or inflammations of the fingers. There are very often complications with neuralgias of other nerves, particularly with cervical, intercostal, and sciatic neuralgia. The course, duration, and termination of cervico-brachial neuralgia are about the same as in other neuralgias.

If injuries of the finger, venesection, or similar injuries have in-

duced brachial neuralgia, neurotomy proves triumphant, particularly in recent cases, and when the neuralgia has not become "habitual." Besides other remedies that we have already mentioned, the internal administration of oil of turpentine has some reputation; we shall refer to it again when speaking of sciatica.

CHAPTER VIII.

INTERCOSTAL NEURALGIA.

We designate as intercostal neuralgia the morbid excitement of one or several spinal nerves, particularly of those which as intercostal nerves pass along the upper intercostal spaces to the sternum, and along the lower spaces to the epigastrium.

Intercostal neuralgia is among the most frequent forms of neuralgia. It is met more frequently in women than in men, shows a surprising predisposition for the sixth, seventh, and eighth intercostal nerves. We have already spoken of the shrewd explanation that *Henle* gives for this peculiarity. The fact that the inferior intercostal nerves are chiefly affected, that is, the ones that empty their blood into the hemiazygos vein, supports the view that one of the chief causes of intercostal neuralgia is dilatation of the venous plexus, which most readily occurs at these places, and exercises pressure on the roots of the spinal nerves. In some cases the dorsal nerves are pressed upon, where they pass through the intervertebral foramina, by inflamed vertebræ, or, after they have passed through, by carious ribs and swollen glands. Intercostal neuralgia not unfrequently occurs after recovery from pleurisy. Within a year I have seen two cases which undoubtedly had this origin. It is doubtful what anatomical changes in the neurilemma or in the parts surrounding the nerves, during a pleuritis or the reabsorption of the pleuritic exudation, have occurred in these cases. The same is true of those cases of intercostal neuralgia that occasionally accompany tuberculosis of the lungs. Lastly, as the disease is particularly liable to occur in hysterical women with chronic uterine difficulty, it has also been referred to a propagation of the morbid excitement from the nerves of the uterus through the spinal medulla to the brachial plexus (*Bassereau*).

In intercostal neuralgia, the three painful points mentioned by *Valleix* are more frequently observed than in most other forms of neuralgia. The first or vertebral point is in the posterior part of the intercostal space, somewhat outward from the spinous process, and about on a level with the point of exit of the nerve from the intervertebral foramen. The second or lateral point lies in the middle of the

intercostal space; it corresponds to the point of division of the intercostal nerve; from it the nerves pass toward the surface. The third point is near the sternum between the costal cartilages in the upper intercostal nerves, in the lower ones it is in the epigastric region somewhat outward from the median line; it is called either the sternal or epigastric point, and corresponds to the place where the terminal branches of the intercostal nerves approach the skin. These usually very circumscribed points are generally so sensitive to pressure, that the patient cries out as soon as they are touched. The patients often discover the points themselves, and voluntarily point out their position to the physician, and, as the pain very much resembles that from a contusion, they are sure they must have struck themselves somehow. This constant pain is increased by deep inspiration, coughing, sneezing, occasionally also by moving the arms, as well as by touching the points; and attacks of lancinating pain are excited, which generally start from the vertebral point and pass anteriorly along the intercostal space; occasionally they pass forward and backward from the lateral point. Hard pressure often relieves the pain. *Romberg* tells of a man whose coat had become threadbare at the point where he was in the habit of pressing his hand. The frequent complication of intercostal neuralgia with herpes zoster is very interesting, although just as obscure as the disturbances of nutrition in the parts supplied by other nerves affected with neuralgia. The disease runs a very irregular course; it usually develops slowly and passes off gradually after variable duration. Occasionally it is very obstinate and hangs on for years.

For the treatment of intercostal neuralgia we would advise the repeated application of blisters to the points douloureux, and particularly the employment of the induced, or, still better, of the constant, current of electricity. During the treatment any anomalies of constitution are to be looked after, and, where the pain is very severe, morphia should be used internally or subcutaneously as a palliative.

CHAPTER IX.

MASTODYNIA—IRRITABLE BREAST (*Cooper*).

It is doubtful whether mastodynia be a neuralgic affection of the branches of the intercostal nerves going to the mammary gland, or of the nervi supraclaviculares anteriores. According to *Romberg's* excellent description, women, about the period of puberty, or from then to the thirtieth year, without any perceptible cause, become sensitive to the slightest touch at one or more points over the mammary

gland. Severe pain, like tic douloureux, occasionally shoots out toward the shoulder, axilla, or hip. Occasionally, at the height of these attacks of pain, vomiting comes on. The patients cannot lie on the affected side; they are unable to bear the weight of the breast. The pain usually increases shortly before the appearance of the menses. The disease often lasts for months or years without any perceptible change in the mammary gland. In other cases, sharply-bounded, very movable tumors, as large as a pea or a hazel-nut, develop in the mamma and form the starting-point for the pain, occasionally they cease to be painful; they consist of connective tissue, not of the substance of the gland; they have been previously mentioned as tubercula dolorosa or neuromata. Fur worn on the breast, a plaster of equal parts of emplas. saponat. and extr. belladonna, recommended by *Cooper*, and pills of ext. conii, ext. papaver (ana gr. ij), ext. stramonii (gr. $\frac{1}{4}$—$\frac{1}{2}$), are mentioned by *Romberg* as the most trustworthy remedies.

CHAPTER X.

LUMBO-ABDOMINAL NEURALGIA.

Neuralgia lumbo-abdominalis is the form that affects the cutaneous nerves of the lumbar plexus, going to the lower part of the back, to the nates, anterior abdominal wall, and genitals. In this neuralgia, also, there are constantly pains at circumscribed points, which are occasionally accompanied by lancinating pains. These points are: 1. The lumbar points, somewhat outward from the first lumbar vertebræ. 2. The hip point, somewhat above the middle of the crest of the ilium, where the ilio-hypogastric nerve perforates the transversalis muscle. 3. The hypogastric point, somewhat inward from the anterior-superior spine, where the ilio-inguinal nerve perforates the transversalis muscle; and, lastly, some points on the mons veneris, vulva, or scrotum at the terminations of the cutaneous nerves. Where the posterior branches are affected, the lancinating pains chiefly pass toward the nates; where the anterior are affected, they chiefly pass toward the external genitals. The etiology, course, and treatment of lumbo-abdominal neuralgia are exactly the same as in intercostal neuralgia.

CHAPTER XI.

NEURALGIA ISCHIADICA—ISCHIAS—ISCHIAS NERVOSA POSTICA COTUNNII—SCIATICA—HIP-GOUT.

By sciatica we mean a neuralgic affection of the sensory nerves of the sciatic plexus, which is formed from the fourth and fifth lumbar and first and second sacral nerves.

Etiology.—The nerves forming the sciatic plexus may be pressed upon by carious or carcinomatous vertebræ, just where they pass through the intervertebral foramen, and may thus be morbidly excited. And sciatica not unfrequently depends on pressure, which acts on the sciatic plexus in the pelvis. In a patient with lymphatic leuchæmia, whom I had the opportunity of observing at Greifswald, a severe intermittent neuralgia, which was undoubtedly caused by the pressure of the immensely-swollen retroperitoneal glands on the sciatic plexus, was for years the most prominent symptom. Tumors in the pelvis occasionally act in the same way, by pressure on the sciatic plexus, particularly ovarian cysts, collections of hard fæces, or, as in a very instructive case of *Bamberger's*, collections of cherry-pits in the sigmoid flexure, also the gravid uterus, particularly if the child's head remain long impacted. Cases of sciatica that are very obstinate, but usually terminate in cure, result from the pressure of exudations of parametritis and perimetritis in the subperitoneal tissue or in capsulated intraperitoneal spaces on the sciatic nerve.

Lastly, irritation of the peripheral branches of the sciatic may sometimes be found as a cause of the sciatica. In this class belong the cases due to pressure from tight boots, from phlebotomy, aneurisms of the arteries of the lower extremity, tumors near the nerve, etc.

Among the causes of sciatica that leave no perceptible anatomical changes are excessive straining, suppression of habitual perspiration and exanthemata, and catching cold. It is quite natural that the latter should occur. Indeed, the majority of cases of sciatica are of rheumatic origin, as they result from exposure to cold of the skin covering the sciatic nerve, as occurs particularly in windy privies. It is not at all astonishing that, among the cutaneous nerves, the trigeminus and sciatic should be affected most frequently; the former being all day exposed to the danger of catching cold, and the latter being exposed for a short time once or twice daily.

Statistics show that sciatica is one of the most frequent forms of neuralgia, that it rarely occurs among children, is most frequent between the ages of twenty and sixty years, and that it occurs more frequently in males and the lower classes than in females and the higher classes.

Symptoms and Course.—Most cases of sciatica support *Romberg's* assertion that there is no cutaneous nerve of the lumbar and sacral plexus, from the hip-joint to the ends of the toes, which may not be affected with neuralgia, and that it is only tradition that locates the pain in the trunk of the nerve. The most frequent seats of the neuralgic pain are the nervus cutaneus femoris posterior, in which

case the posterior and outer part of the thigh becomes painful; the superficial branch of the peroneal nerve, where the pain is in the outer and anterior surface of the leg and dorsum of the foot; the sural nerve (communicans tibialis), where the pain is in the outer side of the ankle and foot. The sensory filaments going to the sole of the foot are rarely the chief seat of pain. In the heel and back part of the sole of the foot severe neuralgia, proceeding from the terminal branches of the tibial nerve, is sometimes seen. *Valleix* gives, as the most frequent points douloureux, some points behind the trochanter, about three points in the thigh corresponding to the course of the chief trunks, some points on the knee, one below the head of the fibula, one above the outer ankle, and a few points on the ankle and dorsum of the foot.

Sciatica rarely begins with great severity; it usually develops gradually and slowly attains its height. The patients are never free from pain, but complain of its constant presence deep in, particularly near the tuberosity of the ischium, at the point where the sciatic nerve passes out, and of pains in the small of the back. As the latter do not come from the branches of the sciatic, but from the posterior sacral nerves, *Romberg* calls them sympathetic. Besides these constant pains, there are convulsive pains, particularly in the course of the above-named nerves. A division into ascending and descending sciatica is made, from the direction of these pains being from below upward or the reverse. The pains sometimes begin spontaneously, especially after going to bed, so that they not unfrequently drive the patient out of bed; sometimes they are caused by outward pressure and movements of the legs. Even moderate tension of the fascia may have this effect; consequently the patient usually lies with his legs slightly flexed. Greater tension of the fascia in coughing, sneezing, or straining, is often accompanied by severe pain. In walking, the patient places the foot of the affected side very carefully, because any quick motion or misstep usually causes severe pain. Participation of the motor filaments in the disease, or a transfer of the irritation from the sensory to the motor filaments through the spinal marrow, not unfrequently causes cramps in the calf or other muscular contractions. Disturbances of nutrition in the parts supplied by the affected nerve are not found in sciatica as they are in other neuralgias. If the disease prove chronic, the limb which the patient favors not unfrequently emaciates decidedly. Occasionally the excitability of the sciatic is gradually impaired, and we have anæsthesia and partial paralysis.

Sciatica is a very obstinate affection. Even in favorable cases several weeks usually elapse before the disease disappears, and it generally subsides as gradually as it developed. In less favorable

cases it often lasts for months or years. Even after the disease has disappeared, relapses very readily occur.

Treatment.—The causal indications can rarely be fulfilled. If disease of the vertebræ be the cause of the sciatica, we may use moxa, the hot iron, etc., to the back. Of the injurious influences that act on the sciatic plexus in the pelvis, overfilling of the sigmoid flexure is almost the only one that is accessible. Although rarely the sole cause of sciatica, this is a frequent complication, hence the old habit of beginning the treatment with a laxative is quite proper. If sciatica remain after difficult delivery, and if we have reason to refer it to a para or perimetritis, we may employ frequent abstractions of blood, and use cataplasms for a long while. In rheumatic sciatica warm-baths are useful, particularly the systematic treatment with baths at Teplitz, Warmbrunn, Wiesbaden, or Wildbad. Among the antirheumatics administered, iodide of potassium in large doses appears to do most good. I saw relief of the pain occur particularly in those cases where the running from the nose, and the eruption caused by the iodine, came on early.

In recent cases, the indications from the disease are best answered by the local abstraction of blood, and wet cups are preferable to leeches. The almost universal mention in text-books of one or more venesections, in the treatment of sciatica, appears to be merely out of respect to old medical authorities, particularly *Cotugno*, for at present no one ever bleeds for sciatica. In cases not very recent we should use derivatives to the skin, particularly blisters. We apply the first blister to the small of the back near the vertebræ, on a level with the points of exit of the nerves forming the sciatic plexus; the second behind the trochanter, and so gradually pass down to the foot, applying blisters to the places where the nerve lies close under the skin. Besides blisters, superficial linear cauterizations of the skin in the course of the nerve, the energetic application of the hot iron and of moxæ to certain points in the course of the nerve, as well as the actual cautery to the dorsum of the foot and between the outer toes, and finally, even the cauterization of the ear, particularly of the helix, have often been tried. After the use of the last-mentioned remedy there is almost always temporary benefit, which it is difficult to explain. Even in the most obstinate cases of sciatica, the induced, or still more the constant, current of electricity rarely fails. Among specific remedies, oil of turpentine as an electuary (ol. terebinth. ʒ i, mell. ℥ i, a tablespoonful twice daily), plays a very important part in the treatment of sciatica. Among others, *Romberg* speaks very highly of it. Neurotomy should only be performed on small branches, in whose peripheral terminations we can clearly locate the starting-point of the morbid

excitement. Regarding the use of veratrine, aconite, and morphine as palliatives, we may refer to what we said in the treatment of neuralgias in general.

CHAPTER XII.

CRURAL NEURALGIA—ISCHIAS ANTICA COTUNNII.

Besides the branches of the lumbar plexus whose neuralgic affections we have described as lumbo-abdominal neuralgia, the sensory filaments going to the thigh and leg are occasionally affected by neuralgia. This affection has received the peculiar name of ischias antica, because in it the pain is along the anterior and inner surface of the thigh, leg, ankle, and dorsum of the foot, and in the great and second toes, instead of being along the outer and posterior surface as in sciatica. Ischias antica, or crural neuralgia, is far more rare than ischias postica. Like the latter, it may be caused by pressure on the nerves at their point of escape from the spinal canal, or by injuries to the sacral plexus in the pelvis, or to its peripheral branches. Irreducible inguinal hernia, sprains of the thigh, hip-joint disease, not unfrequently induce morbid excitement of the cutaneous branches by pressure on or tension of the crural nerve. The course and results of crural neuralgia are analogous to those of sciatica, and the treatment must be according to the same general laws.

In very rare cases the obturator nerve is also affected with neuralgia. This is characterized by the extension of the pain to the inner surface of the thigh, and, as the motor filaments of the obturator supplying the adductor muscles usually participate in the morbid excitement, the function of these muscles is frequently disturbed. The sudden occurrence of these symptoms, together with those of acute obstruction of the intestines, with ileus and peritonitis, are the only means of diagnosticating strangulated hernia through the obturator foramen.

CHAPTER XIII.

ANÆSTHESIA OF THE CUTANEOUS NERVES.

Anæsthesia—that is to say, lack of sensitiveness to external impressions—arises:

1. When the portion of the brain by which the excitement of sensory nerves is recognized has been destroyed or incapacitated. In such a case, notwithstanding that the patient is entirely insensible to external impressions, the excitability of the sensory nerves may still be normal. For the present, we shall, without notice, pass over this

form of anæsthesia, which we have mentioned as one of the symptoms of many of the diseases of the brain, as this section of the work is to be devoted to a study of diseases of the peripheral nerves.

2. Anæsthesia may occur when, owing to destruction of the conducting fibres of the spinal cord, transmission of the impressions from the peripheral nerves to the brain is interrupted. In this form, likewise, the excitability of the peripheral nerves may be normal. In treating of myelitis, we have already mentioned that, while the stronger stimulants, acting upon the periphery, fail to call forth any excitement at the centre, even a very feeble stimulus is frequently transmitted from the sensory to the motor fibres, and gives rise to reflex symptoms. Such phenomena as these prove that the excitability, both of the motor and sensory nerves, still remains unimpaired below the point at which the conducting power is interrupted. This form of anæsthesia, also, is not the subject of the present section.

3. Anæsthesia occurs when there is nutritive disorder capable of destroying the irritability of the peripheral nerves, as well as when their connection with the brain and spinal cord has been interrupted mechanically. It is with this form alone that we are at present concerned. We shall here observe that we consider all sensory and motor nerves as peripheral as soon as they leave the brain, no matter whether they continue to lie within the skull or spinal canal, or whether their track be outside of the same. This is by no means an arbitrary distinction. It depends chiefly upon the fact that the portion of nerve lying within the skull or spinal canal is in just the same state as the peripheral portions, as regards extinction of its irritability upon interruption of its connection with the central organs. In the motor nerves this can be proved with ease. In a patient who is unable to move the right side of his face at will, owing to an apoplexy of the left corpus striatum, the nerves of the palsied side retain their irritability for weeks, and all the muscles of that side may be made to contract under the influence of the induced electric current. On the other hand, if the facial be destroyed within the skull after its departure from the brain, its irritability soon becomes extinct, just as though its peripheral branches had been divided, and it is impossible to make the muscles of the palsied side contract by the influence of the induced current. With regard to the sensitive nerves, the proof cannot be made so plain, yet we are warranted in inferring that the conditions are very similar. At all events, in central anæsthesia, the trigeminus long remains sensitive, as is proved by the continuance of the power of reflex action, for instance, as is shown in winking when the conjunctiva is touched.

Etiology.—The nutritive disorder, whereby a nerve is deprived of

its irritability, is often so subtle in character as to escape our direct observation. The moment a nerve loses its supply of arterial oxygenated blood, it loses its functional power. There is no doubt but that this loss of irritability is due to physical or to chemical changes within the nerve. Not unfrequently, in the region of an artery occluded by an embolus or thrombosis, we find anæsthesia to exist until the collateral circulation has been established. The action of continued cold induces anæsthesia of the skin. Part of this is undoubtedly due to the contractile power of cold upon the skin, and to the anæmia of the cutaneous nerves thus produced. Of the anæsthesiæ of peripheral origin—that is, depending upon extinguished irritability of peripheral nerves, without appreciable alteration of their structure—there is the so-called rheumatic anæsthesia, which not unfrequently takes place after exposure to cold, as well as a form which *Romberg* has noticed in the hands and forearms of washerwomen. It is doubtful whether the anæsthesia arising from lead-poisoning be due to impalpable nutritive lesion of the peripheral nerves, or to a morbid state of the central organs. This is also true of the locally anæsthetic action imputed to ether and chloroform. It does not seem to me to be proved that the action of these substances is purely local, when locally applied; and I think it more likely that their influence is more or less centric at such times, if not entirely so. Can we expect to find a perfect anæsthetic action from such agents, the cerebral function remaining intact meantime? It bears a peculiar relation to hysterical anæsthesia, of which we shall treat in our chapter upon hysteria.

Anæsthesia due to permanent and uniform pressure of an overtight bandage or piece of clothing, constitutes a connecting link with those forms of anæsthesia where structural lesion of the peripheral nerves is not merely a matter of supposition, but can be absolutely demonstrated. In such cases we find all the tissues, subjected to pressure, to be in a state of atrophy and fatty degeneration, and, of course, the peripheral nerves are by no means exempt. The irritability of a nerve is not unfrequently suspended, and its continuity is finally interrupted by the pressure of a tumor, an exudation, or extravasation. In other cases of anæsthesia, portions of the nerve are destroyed by inflammation. It would be superfluous further to continue the enumeration of the structural alterations capable of producing anæsthesia, as it would be but a repetition of what we have already stated in treating of the etiology of neuralgia. The very causes which, excited mildly, or for a short time, serve to exalt the excitability of a nerve, have a more pernicious effect should their action be prolonged or intensified, as the nervous excitability is then arrested, or even the nerve itself may be destroyed.

Symptoms and Course.—Anæsthesia is either complete or incomplete. In the first case, irritants acting from without, especially pressure and change of temperature, produce no sensation whatever. In the second case, though an impression is produced, it is indistinct and inaccurate. Where the anæsthesia is incomplete, there is often a numb or muffled sensation. The patient feels as though a foreign body lay between his skin and the object which he actually touches. *Henle* believes that this state of affairs is due to an extinction of sensibility of the peripheral terminations of the nerves, and a retrocession of the sensibility in the direction of the centre, so that there actually is a layer of passive substance between the nerve and the object touched. Upon this hypothesis, it is very easy to explain why cold, whose immediate action is upon the tips of the sensory nerves, should make the skin feel numb and muffled. There is a peculiar form of anæsthesia called *analgesia.* In these cases there is no pain, even under the most intense irritation of the nerve, although the sense of touch is not impaired. We have no satisfactory means of accounting for analgesia. With *Henle*, we might suppose it to be a low grade of anæsthesia, and might assume that excessive irritation, instead of producing the usual amount of perception, namely pain, merely gives rise to that normally induced by moderate stimulation of the nerve, were it not that, under such a condition, moderate irritation should be imperceptible, which is not the case in analgesia. The modifications of anæsthesia, known as partial losses of sensation, are still more obscure. Here the effect of certain irritants is extinguished, while that of other irritants of no greater power continues. For instance, there may be a loss of the sense of touch, with unimpaired sense of temperature, or conversely.

The so-called "law of excentric impression," according to which the excitement of a sensory nerve, even though produced by irritation of its trunk, always produces the impression of an excitement of its peripheral extremity, fully explains why patients often suffer pain in regions which are entirely insensible to external irritation, a phenomenon to which the name of *anæsthesia dolorosa* has been applied. Anæsthesia dolorosa arises, first, in all cases in which a nerve has lost its sensitiveness throughout a certain portion of its length, while, at the same time, a severe irritation is made to act, above this point, upon that portion of the nerve which still remains in connection with the brain. It is easy to see that both of these factors often coexist, as when a tumor presses upon a nerve. Here the tract of nerve, lying between the compressing growth and the brain, remains excitable and receptive of lasting impressions, while from the tumor to the periphery the nerve has lost its irritability.

In anæsthesia of the muscular nerves, perception of the degree of contraction or relaxation of the muscles is diminished or destroyed. Sometimes such patients are capable of making every motion required of them, but are unable to tell, without the help of vision, to what extent they have moved. They can hold fast to an object as long as their eyes serve them as "check-lines of motion" (*Romberg*), but let it fall as soon as their eyes are closed. If anæsthesia of the muscular nerves affect the lower extremities, the patient is often able to move about tolerably well during the day, but in the dark he will stagger about, and is quite unable to walk even a step or two (see chapter on tabes, page 305).

Very often anæsthesia is complicated with derangement of circulation and nutrition in the insensible region. The temperature of the part is often depressed several degrees, its function is impaired, and its secretions are diminished. Besides this, there is an increased vulnerability of the region. A comparatively trifling degree of heat or cold suffices to burn or freeze the part. Wounds and ulcers heal more slowly, or not at all; bed-sores are very apt to form. The epidermis and nails become fissured and liable to scale off; the skin becomes livid, and œdema develops in the subcutaneous areolar tissue. This peculiar state of the insensible region is in some degree attributable to the retardation of the circulation, but the cause of this retardation is obscure. From experiments made by *Axmann*, it seems probable that it is due to an implication of the sympathetic fibres which accompany the spinal nerves. In frogs, whose spinal nerves had been divided between the spinal marrow and the spinal ganglion, *Axmann* only found anæsthesia and palsy, but no nutritive disorder of the palsied part. If, however, the nerves were divided at a point beyond that at which they are joined by the communicating branches of the sympathetic, in addition to the anæsthesia and palsy, there was desquamation of the epidermis, obstruction of the capillary circulation, and œdema.

If the nerve, which has been cut off from its central organ, or which has lost its irritability from any other cause, contain both motor and sensory fibres, partial anæsthesia and partial palsy are combined. It would almost seem as if a nerve which had lost its irritability could transmit this passive condition through its ganglion to other nerves, just as an excited nerve can communicate its excitement by means of its ganglion-cells to other nerves. It is in this way that we account for the impairment of the sense of taste and smell in anæsthesia of the trigeminus, and for the anæsthesia of the auditory organs in cases of nervous deafness, as well as for the partial paralysis which sometimes supervenes upon long-continued anæsthesia.

It is not always easy to distinguish peripheral from central anæs-

thesia. We may rely for a diagnosis, in some degree, upon the following points: 1. Upon the extension of the anæsthesia and its complication or non-complication with motor palsy. If the anæsthesia be confined to one side, and be combined with hemiplegia, it is probably of the cerebral form. If, however, it extend over the lower half of the body, and be combined with paraplegia, the case is almost always one of disease of the spinal marrow. The coexistence of palsy of the face with facial anæsthesia indicates a central cause of disease, while anæsthesia, with normal motion, indicates its peripheral origin. 2. The character of the reflex symptoms are of great importance as a means of distinguishing between the two forms. When the anæsthesia is peripheral, the nerve being either separated from the brain, or having lost its power of excitability, no stimulus can be transmitted from it to the motor nerves, and there can be no reflex symptoms; but if the anæsthesia be central, that is, if the region of the brain in which we become conscious of impressions be disabled, the route by which the excitement is transmitted from sensory to motor fibres may still remain quite perfect, and reflex symptoms may readily appear. It is just the same, in cases of circumscribed disease of the spinal marrow, as in spondylarthrocasis, and partial myelitis. On the other hand, in diffuse inflammation, as in tabes dorsualis, reflex symptoms are not apt to be seen. The following is a striking example of the difference of these two conditions. When anæsthesia of the conjunctiva occurs from apoplexy, the eye may be touched without the patient's perceiving it, and yet he involuntarily closes the eye because a stimulus is transmitted from the sensory fibres of the trigeminus to the motor ones of the facial. But if the anæsthesia be dependent upon loss of the ganglion of Gasser, or of the ramus-ophthalmicus, the patient neither feels the touch, nor does he involuntarily close his eye, since transmission of the excitement to the facial has then become impossible.

The course of an anæsthesia depends upon the main disease. If a nerve be divided by an incision, sensation is sometimes restored after a while. If, however, a large portion of a nerve have been destroyed, the anæsthesia will remain during life. Rheumatic anæsthesia, as well as that occasioned by a moderate and uniform pressure over the course of the entire nerve, warrants a better prognosis than the other forms.

Treatment.—The treatment of anæsthesia is never satisfactory, excepting when it is possible to remove the cause. Sometimes a surgical operation is indicated, sometimes a course of antirheumatic treatment. It does not always immediately happen that cessation of the cause is followed by removal of the effect. In such cases spirituous and ethereal embrocations may be used, as well as eleo

tricity, cold douches, the baths of Gastein, Wildbad, or Pfäffers, and even the disgusting so-called Thierbäder. The reëstablishment of extinguished sensibility of sensory nerves by such means, however, is a very uncertain matter.

CHAPTER XIV.

ANÆSTHESIA OF THE TRIGEMINUS.

Etiology.—According to an explanation given in a previous chapter, the same pernicious agents which we have found to be the causes of prosopalgia, when of greater severity or of longer duration, may give rise to anæsthesia over the region of the trigeminus. In anæsthesia of one-half of the face, the Gasserian ganglion of that side has been found compressed, degenerate, or destroyed by a tumor or inflammatory process. In other cases but one of the three branches has suffered permanent pressure or other injury within the skull, or else in the passage through the sphenoid bone. In still others, which indeed are the most common of all, a few twigs only of the trigeminus have been cut through, or have been destroyed by the pressure or suppuration of glands or tumors, or torn during the extraction of teeth (especially the inferior alveolar branch). The favorable course taken by some cases shows that anæsthesia of the trigeminus may also proceed from transitory affections of the neurilemma, or of the nerve itself, which probably are due to cold.

Symptoms and Course.—When all the fibres of the trigeminus have lost their irritability, it may be assumed that its main trunk or the ganglion of Gasser is degenerated or destroyed. In such cases the face is divided into halves, standing in abrupt contrast with one another, the one being sensitive, the other insensible. The various cavities of the face, whose supply of nerves comes from the trigeminal, are in similar condition. Upon the affected side the eye may be touched, the bulb pinched or pricked, the nose irritated with a sharp instrument, with snuff, or with acrid vapors, without the patient's feeling it. If the patient put a glass to his lips, he feels but one side of it, and receives the impression that it is broken. Half of the tongue also is insensible. The saliva flows from the mouth at the affected side, and the remains of food hang to the lips without the patient's being aware of it. Upon irritating the conjunctiva, there are none of the reflex movements referred to in the previous chapter. If, however, the eye be exposed to a strong light, reflex action occurs, as irritation of the optic nerve transmits its impression to the facial nerve Not only do the patients fail to wink when the conjunctiva is touched

but they do not sneeze when the nasal mucous membrane of the affected side is irritated, since no reflex action upon the respiratory apparatus can be set up in this region. Nutritive disorders of the affected side are associated with these symptoms, similar to those which may be provoked artificially in the lower animals by extirpation of the ganglion of Gasser, such as varicosities of the eye, with ulceration and atrophy, fungous flabbiness of the gums, bleeding from the mouth and nasal cavities, blueness and œdema of the cheeks. In contrast to the observed cases of "ageustia," there are a good many others in which the sense of taste remained normal. The sense of smell and of vision often suffers; but in such cases it is difficult to say what part the disorders of the nasal mucous membrane and bulb above referred to play in producing such impairment of function. If both portio major and portio minor of the trigeminus be destroyed, in addition to the palsy of sensation of one side of the face, there will be palsy of the muscles of mastication upon that side. If the destruction of the ganglion be caused by the presence of a large tumor or other extensive disease affecting the base of the skull, the function of other cerebral nerves is likewise usually disturbed, especially that of the oculomotorius, the facial, the acoustic, and there are ptosis, dilatation of the pupil, palsy of the corresponding side of the face, and deafness of the ear of that side.

If but one branch of the trigeminus, instead of the whole nerve, be diseased, the anæsthesia is limited to the region of the face which is supplied by that branch; if it be the first branch, to the socket of the eye, the second to the nose, the third to the cavity of the mouth. If the affection be confined to a few twigs alone, the insensible parts of the face are still smaller, and the cavities may be normal.

An isolated central anæsthesia of the trigeminus without, simultaneous anæsthesia of the corresponding half of the body, is one of the greatest of rarities. In a previous chapter we have stated the chief means of distinguishing such a case from a peripheral anæsthesia.

With regard to treatment, we must also refer to what has already been said of the treatment of anæsthesia in general.

SPASM INVOLVING PARTICULAR PERIPHERAL NERVES.

In the following chapter we shall treat of the morbid conditions of excitement to which the various motor nerves are subject. Just as excitement of a nerve of sensation is evinced by a feeling of pain and by reflex symptoms, so that of a motor nerve is indicated by contraction of the muscle which it supplies. If the motor nerve be acted upon by

a series of morbid impressions in quick succession, the muscular contraction resulting from the first impression will continue until the second one succeed it. In this way permanent muscular contractions occur which are called *tonic* spasms. If the nerve be irritated at somewhat longer intervals, so that the muscle relaxes ere another impression make it contract again, thus causing alternate spasm and relaxation, we speak of *clonic* spasms.

In the healthy subject the motor nerves receive a portion of their stimulus from the grand centre of the will, thus producing the voluntary movements; another portion of it is obtained from parts of the brain, which, when thrown into excitement, are independent of the action of the will. To this class belong the movements which occur during the painful emotions, and in anger, and which we call outbursts of pain and anger. It is not through our will that we contort the face in weeping, when we are in grief; we do not voluntarily clinch the fists when angry; these events occur in spite of our will. Finally, in the healthy subject, there is another series of motions which are likewise brought about without the action of the will, and even against the will, by the transmission of a centripetal impression to a centrifugal channel. These are called normal reflex movements; and a distinction is made between those induced by a regularly recurring stimulus of moderate intensity, such as the respiratory movement which is called forth by the collection of carbonic acid in the blood, and the act of deglutition which follows upon the introduction of a bolus into the œsophagus (these being called *automatic* movements), and those induced by stronger but less frequent impressions, such as sneezing, winking, and the like.

The terms cramp, spasm, and hypercinesis, are applied to certain morbid conditions in which excitement of the motor nerves is produced by an irritant of unusual and generally speaking of unknown origin, and entirely independently of the volition, or else to a condition in which the action of an ordinary stimulus calls forth an excitement of extraordinary extent and violence in the motor tract.

It is impossible in the present state of science to classify spasms according to the kind of irritant which gives rise to the nervous excitement, or according to the region in which the irritant operates. Spasms of which we are about to treat are the result of a variety of causes, and often of unknown causes; and although in some instances the affection is confined to the province of a single motor nerve, yet we are not at all certain that it proceeds from direct irritation of the fibres of that nerve.

CHAPTER XV.

SPASM OF THE FACIAL NERVE—MIMIC SPASM OF THE FACE—TIC CONVULSIF.

ETIOLOGY.—A morbid irritability involving the twigs of the facial nerve which go to the muscles of the face, but which does not involve the fibres running to the stylohyoid and digastric muscles, is called *tic convulsif* whenever it is not combined with general convulsions. The cause of this affection of the facial is obscure. Probably the cases of isolated irritation to which alone we refer at present are never dependent upon lesions of the brain. Nor does the cause of tic convulsif seem ever to be an irritation of the facial within the skull, or during its passage through the canal of Fallopius. Whenever the morbid excitement of this nerve is due to the action of some noxious influence upon the face itself, such as cold, a contusion, or the pressure of a tumor, it becomes a matter of doubt whether these influences act directly upon the fibres of the facial nerve, or whether their immediate effect is not upon the filaments of the trigeminus, the facial merely becoming irritated by reflex action. Sometimes, too, tic convulsif has been viewed as a reflex neurosis, arising from the irritation of remote organs, such as the rectum in helminthiasis, or the uterus in hysteria. Finally, mental emotion and the imitative instinct are set down among the causes of tic convulsif. The affection seems to be somewhat more common among men than among women.

SYMPTOMS AND COURSE.—In almost all cases of tic convulsif, but one side of the face is affected; sometimes the spasm is clonic, sometimes tonic. *Romberg*, in a few brief but graphic lines, gives the following description of the mimic spasm: "Grimaces occur, either intermittent or constant, involving one side of the face, and, more rarely, both sides. In the former case they consist chiefly of elevation or depression of the occipito-frontal muscle, corrugation of the eyebrows, blinking and closure of the eyelids, twitching and snuffling of the alæ nasi, and drawing up and down of the corners of the mouth. These attacks set in suddenly, and as suddenly subside, to recur, with equal suddenness, at short intervals. In permanent tonic contraction of the facial muscles, the furrows and hollows in the affected side of the face are deeper; the tip of the nose, the commissure of the lips, and the chin are drawn toward the convulsed side. The muscles feel hard and tense, and so impede motion that the one eye cannot be as completely closed as the other." The patient is unable to prevent or to control these motions at will. As a rule, any individual motion, made voluntarily, is accompanied by involuntary contractions of other muscles.

In the beginning of the disease the affected side of the face is often somewhat painful. Afterward the pain abates, and, as a great rarity it is followed by incomplete anæsthesia. In some cases, instead of all the nerve-fibres of the facial, a few only are involved in the morbid irritability, as the palpebral branch, the malar, or the labial, or, as in a case reported by *Romberg*, the auricular branch. The affection of the rami palpebrales causes a series of rapid openings and shuttings of the eye—*nictitatio*, or a rigid, violent closure of the lids—*blepharospasmus*. The derangement of the buccal and labial branches gives rise to a movement of the lips, upon one or both sides, as if from laughter, the so-called *risus caninus*, or *sardonicus*. In the case of tic convulsif of the auricular branches, described by *Romberg*, repeated fits of twitching of the ear set in daily, during which the ears were drawn up and down for fifteen minutes at a time. Sometimes the tic convulsif spreads to other nervous trunks, as does the tic douloureux; so that movement is excited in the muscles of mastication, or those in the region of the hypoglossal nerve, and of the accessory of *Willis*, or of some of the spinal nerves.

The course of the disease is usually chronic, and of uncertain duration. Commencing suddenly (or gradually, which is rather more common), it generally continues during the remainder of life. In order not to mistake the tonic form of mimic facial spasm for the distortion of the face arising from hemiplegia, we must observe the unimpaired muscular power of the undistorted side, and the normal behavior of its muscles under the induced current. Moreover, according to *Bruns*, in hemiplegic palsy, upon handling the healthy side of the face, we do not find the smallest trace of permanent contraction of the muscles, and, by stroking and drawing upon the skin of the face, the mouth may be brought into proper position.

Treatment.—In recent cases of tic convulsif, a diaphoretic and derivative procedure always seems to do good, while, according to my experience, an old case resists all treatment, even the application of electricity. Where the morbid irritation of the facial manifestly is of reflex origin, and if we can tell from which filaments of the trigeminus the disturbance proceeds, division of those twigs from the brain by neurotomy is indicated. In two cases reported by *Romberg*, where the supra-orbital nerve was divided, great benefit was obtained. On the other hand, division of branches of the facial nerve is not advisable, as that procedure gives rise to another deformity scarcely less unpleasant than the former one, namely, mimic facial palsy. In one obstinate case of tic convulsif, subcutaneous incision of the facial muscles has been practised by *Dieffenbach*, with good result.

CHAPTER XVI.

SPASM IN THE REGION OF THE SPINAL ACCESSORY NERVE OF WILLIS.

Etiology.—Like the facial, the accessory nerve of *Willis* is sometimes the seat of morbid irritability. The pathogeny and etiology of this condition are as obscure as are those of tic convulsif. Violent twisting of the neck, cold, disease of the cervical vertebræ, have been the assigned causes. The nodding spasm, or salaam convulsions, seen in children, particularly at the period of dentition, does not seem to be a genuine affection of the spinal accessory nerve and the cervical nerves. Indeed, from its complications and course, it seems probable that it is symptomatic of cerebral disease, or of an eclampsia.

Symptoms and Course.—Morbid excitement of the spinal accessory nerve is manifested by spasm, either of a tonic or clonic character, in the muscles supplied by that nerve, the trapezius, and the sterno-cleido-mastoideus.

In the former case, in each paroxysm, the head is drawn obliquely downward several times in succession, the occiput approaching the shoulder, and the ear the clavicle. The direction assumed by the head is more forward or backward, according as the contractions are greater in the trapezius or sterno-cleido-mastoideus. In the same way the shoulder-blades and shoulders are drawn up during the contractions of the trapezius. If the irritation spread to the facial, the face twitches also; if it involve the portio minor of the trigeminus, the jaws are convulsively set. If the cervical nerves be also implicated, the head is twisted, and the arms are thrown into convulsive agitation. Such paroxysms, which are usually accompanied by pain in the muscles or their attachments, usually last but for a second or two. At the outset of the disease the attacks are less frequent; as it advances, they recur oftener; so that as many paroxysms as thirty may occur in a minute, driving the patient almost to desperation (*Hasse*). They do not come on during sleep. These clonic spasms of the region of the accessory nerve generally develop in a very slow and gradual manner. They rarely cease, but, as a rule, endure through life, without endangering it.

The term "salaam convulsion" of children is applied to solitary or periodically-recurring paroxysms of clonic spasm of both sterno-mastoid muscles, whereby the child is made to nod its head incessantly, like a Chinese image. The nodding is very rapid, and sometimes increases in rapidity as the attack goes on, making occasionally eighty to a hundred nods a minute. Generally speaking, the muscles of the face, especially the orbicularis palpebrarum, sympathize in the movement. Epilepsy and idiocy develop in some children affected

in this way, while in others it is said to disappear when dentition is complete.

Tonic spasm of the accessory nerve of *Willis* is the cause of the spastic form of torticollis or caput obstipum. The spasm is usually confined to the sterno-cleido-mastoideus, so that the head is inclined forward and downward. The affection occurs more frequently in children than in adults, and perhaps, too, it sometimes affects the fœtus if congenital torticollis be attributable to an intra-uterine fœtal tonic spasm of this muscle. Sometimes the tonic spasm is preceded by a brief attack of clonic convulsions; more generally, however, the muscular contraction is of a tonic character from the first. At the commencement the disease is apt to be considered of little moment, and, as it is usually attended by pain, is looked upon as of rheumatic character. An embrocation of opodeldoc is ordered for the "stiff neck," or else some other equally harmless prescription. Finally, however, the obstinacy of the attack and its gradual aggravation furnish evidence of its more serious nature. The head is drawn more and more to one side, the sterno-mastoid muscle of that side, particularly its sternal portion, stands out like a hard cord, while upon the upturned side of the neck the skin is stretched and the muscles are not prominent at all. If the disease last long, the face becomes distorted, the hypertrophied muscles drawing their corresponding half of the face downward. In the same way the permanent obliquity of the attitude often leads to curvature of the spine, and to sinking of the thorax of the affected side.

Treatment.—Treatment of clonic spasm of the spinal accessory nerve is seldom very successful. It is true that *Moritz Meyer*, in five sittings, completely cured a soldier affected with tic convulsif and clonic spasm of the neck, by faradizing each one of the contracted muscles. The negative results, however, which I myself as well as others have witnessed in the treatment of tic convulsif, by faradization and galvanism, would imply that such brilliant cures were exceptional. So, too, the internal use of sulphate of zinc, and of carbonate of iron, and the application of moxas to the back of the neck, would seem to have sometimes been of service. Section of nerves has not succeeded, although, in two cases, division of the muscles was successful. In a third case, in which the division of the muscles was practised more than once, no benefit was derived.

We can give no rules for the treatment of the salaam convulsion, since the nature of this affection, and especially its etiology, is entirely unknown. The treatment of tonic spasm, which belongs to the province of orthopedic surgery, can boast of some success in cases which were not of too long standing.

CHAPTER XVII.

SCRIVENERS' SPASM—MOGIGRAPHIA.

Etiology.—The term scriveners' spasm is employed to denote a morbid excitement of the motor fibres of the nerves which supply the muscles of the figers, and which renders further writing impossible, by inducing spasmodic contraction of the muscles to which they belong. If such spasms are provoked by other kinds of manipulation, and not by writing, for instance, by shoemaking or by milking, they receive other names, such as "cobblers'" or "milkers' spasm." Numerous as are the hypotheses as to its origin, we know nothing positive as to the pathogeny of this disease, which is not at all uncommon, and which often deprives the individual afflicted of his means of subsistence. The theory of *Fritz* is in some respects satisfactory. He regards it as a reflex neurosis, in which, however, excitement of the motor nerves is not derived from the cutaneous nerves, as in most reflex neuroses, but proceeds from the muscular nerves. The observation that the touch of a feather or paper is not of itself sufficient to excite the cramp, although holding the hand in the attitude of writing, even though it touch neither pen nor paper, will do so, argues in favor of this view. The theory is equally probable, however, that the morbid irritation is not reflected from the sensory to the motor nerves, but proceeds from transmission of the impression of the nerves excited by the will to other motor nerves. Scriveners' cramp would then resemble the convulsive movements of chorea, and stammering, and the movements which disturb the writer would have to be regarded as sympathetic movements.

With regard to its etiology, it is to be observed that the affection is much more common among men than among women, in whom it has only been observed occasionally. Will it not turn out upon examination that, just as in many other diseases (although perhaps less strikingly so), the apparent difference of tendency to the disease in the two sexes is really due to a difference of habit and occupation? Writers' cramp is most liable to occur between the ages of thirty and fifty years, that is, during the time of life when occupation is most intense. Professional penmen, clerks, teachers, and merchants are most subject to the affection. Narrow coat-sleeves, which compress the muscles of the arm during writing, an inconvenient attitude, but, above all (since scriveners' spasm has only come into notice since the introduction of steel pens), the use of a hard steel pen seems to favor the development of the affection. Why the disease should develop under these causes alone is unknown.

Symptoms and Course.—The usual precursory signs of writers'

cramp are a feeling of slight fatigue of the hand, and a sense of insecurity as if the writer must grasp his pen more firmly. Sometimes the affection does not advance beyond this stage. When the disease is fully developed, the muscles of the first three fingers become convulsed during writing, and, in bad cases, upon every attempt to write, sometimes the flexor muscles, sometimes the extensors, are the most contracted. Various forms of this spasm have been recognized according to the predominance of one or other convulsive movement, such as convulsive flexion of the thumb, jerking of the index-finger, loss of control of the hand, and a form made up from the last two of these varieties. During these spasms the pen is rapidly twitched up and down, but, instead of regular, distinct letters, it forms grotesque interrupted scribbling. The occurrence of the paroxysm is favored by apprehensive attention to the subject and fear of its arising. The harder the patient tries to continue his writing, so much the stronger is the spasm, as so much the more is it apt to extend up the forearm and arm. It is usually painless, although sometimes it is attended by a feeling of tension in the arm. The moment the patient suspends his attempt to write, the cramp ceases, and he can execute all other movements undisturbed. In a case of this kind, described by *Romberg*, a nailsmith, as soon as he grasped his hammer and prepared to strike, was seized by painful spasm of the forearm. He was obliged to give up his trade, and afterward become a useful and skilled painter. Writers' spasm is a very obstinate and tedious disorder. Recovery is very rare, and its uniform persistence is hardly ever broken even by a temporary improvement of the symptoms. Some patients, who are ready for any sacrifice in order to be rid of their burdensome disease, and from their anxiety for this means of subsistence, learn to write with the left hand. Unfortunately, however, sometimes in such cases the left hand is also attacked by the disease.

Treatment.—The treatment of scriveners' spasm is as a rule unsuccessful. Experience has shown that very little benefit is to be derived from the long-continued prohibition of writing, and by only allowing the patient to resume the practice cautiously, and only permitting him to use goose-quills, or by the use of cold douches, sea-baths, and stimulating friction. Division of the nerves is of no benefit to the patient, as it causes paralysis of the finger. In one case only has section of the muscles been followed by benefit. In all other instances there was either no improvement at all, or it was a merely transient one. In one case of scriveners' spasm, when, without hoping for success, I applied the constant galvanic current in a manner almost rude, I have succeeded in curing the disease, so that the patient, who for years had written with the left hand, now uses his right once more. The

treatment proposed by *Benedikt*, by which he has obtained some success, and which consists in the application of "spinal-root, and spinal nerve-streams," has utterly failed in my hands; and in one or two cases it has aggravated the disease. If the pathogeny of scriveners' spasm be really as I have suggested above, my treatment of it was an entirely rational one, although I myself was not aware of it at the time. I applied the current to the muscle of the thumb and index-finger, and hence to the sensory muscular nerves running into them: thus if the explanation be correct, the cure would be accounted for by an abatement of the morbid nutritive state, and of the morbid excitability of the sensory nerves of the muscles, from whose reflex action the cramp proceeds, by the catalytic action of the constant current. Sometimes apparatus, by means of which the patient writes without the aid of his fingers, also are of service for a while.

CHAPTER XVIII.

IDIOPATHIC CRAMP OF THE MUSCLES OF THE EXTREMITIES—ARTHROGRYPOSIS.

Etiology.—According to the example of the French authors, the term idiopathic spasm (spasmes musculaires idiopathiques) is applied to tonic contractions which sometimes attack the muscles of the extremities, without being attributable to disease of the brain or spinal marrow. They are analogous to the neuralgias, but we are still less able to point out the anatomical cause of this morbid condition of the motor nerve, causing idiopathic muscular spasm, than to discover that which acts upon the sensory nerves in neuralgia. The harmless course usually taken by the disease makes it probable that idiopathic cramps of the extremities are due to trifling and transient lesions of the nerves and their sheaths. By many observers, these affections are regarded as a form of rheumatism, and are attributed to hyperæmia and œdema of the neurilemma. In some cases, this may be the truth, especially in such as occur in children previously healthy. However, the occurrence of idiopathic muscular spasm during the course of and convalescence from acute and chronic disorders, which have an exceedingly pernicious effect upon the assimilation and nutrition of the body, and which often lead to great functional disturbance, such as typhus, intermittent, Bright's disease, and epidemic diphtheritis, makes it quite probable that in other instances idiopathic muscular cramps are the result of derangement of the tissues, the character of which is almost unknown, and which develops during the diseases above mentioned, giving rise to a great variety of functional disturbance.

The muscular spasms which arise during pregnancy, during labor

and after it, must be regarded as of this character. The affection is most common during childhood, especially the primitive form induced by cold. The statement of *Valleix*, that the disease is so rare, that few physicians ever see it, strikes me as strange, as not only have I myself observed a great number of such cases, but I well remember that *Krukenberg* used to describe it as a by no means uncommon form of rheumatism among children.

Symptoms and Course.—Sometimes the disease is preceded by a feeling of illness, languor, and depression of several days' duration. The actual commencement of the disease is marked by pain, which apparently shoots along the courses of the nerves, sometimes involving the upper and lower extremities simultaneously, sometimes only the hands and forearms, sometimes the feet and legs. In addition to this, there is a sense of formication, and of stiffness and sluggishness in the suffering members. These symptoms having lasted for a variable period of time, fugitive cramps, in the calves of the legs and other muscles, set in, which soon are converted into continued tonic contractions. The upper extremities are usually brought into a state of permanent flexion, the lower into permanent extension. If we are unacquainted with the malady, and see a child thus affected for the first time, we shall be deeply impressed by the aspect of the rigid immovable limbs, the extended knees, the heels drawn up, the thumbs forced into the palms of the hands, and shall be inclined to ascribe it to some serious lesion of a central organ. The attempt to extend the upper extremity or to flex the lower is extremely painful to the patient. The contracted muscles are hard and prominent. Generally the joints seem somewhat swollen by a slight œdema of the skin. Sometimes the tonic contractions extend to the muscles of the back and belly, and even to those of mastication and to those of the face. The affection is either entirely free from fever or else the fever is of but little intensity. In some patients the commencement of the contraction is accompanied by a sensation of oppression and of rush of blood to the head. In many instances the course of the disease is brief; the contractions ceasing after a lapse of a few days, and free mobility becoming reëstablished. In other instances it is more protracted. Sometimes, too, relapses occur after some days or even some weeks have passed. Certain observers, as *Delpeche* and *Hasse*, state that the disease consists of a series of spasmodic attacks, that these paroxysms last for some minutes, some hours, or a day or more, and that in the intervening period there is merely a certain degree of stiffness and swelling of the limbs, accompanied, perhaps, by anæsthesia of the skin and muscles. In the cases which I have had opportunity to observe, no such paroxysms and intervals were discoverable.

Treatment.—The course of the disease, which usually is favorable, renders active therapeutic interference superfluous. *Krukenberg* used to recommend fumigation with juniper-berries. The pain in the limbs and the contractions of the muscles certainly subside quite as soon under this simple treatment as under the use of other stimulating and antispasmodic embrocations, and by the internal use of remedies for convulsions, such as flores zinci, henbane, opium, and the like. If the idiopathic muscular spasm be the effect of some grave general disease, the case is different. The spasms do not then cease until the normal assimilation and nutrition have once more become reëstablished; and treatment must be regulated accordingly.

CHAPTER XIX.

PERIPHERAL PALSY.

The term palsy—acinesis—of the province of the cerebro-spinal nerves is applied to a morbid condition, in which the motor fibres are no longer acted upon by volition, so that the muscles cannot be made to contract at will. Derangements of the voluntary motion having another origin, especially those caused by disease of the bones and joints, are not counted as palsy. Myopathic palsy shall be treated of hereafter.

In treating of disease of the brain, we have already described that class of paralysis proceeding from destruction or derangement of the grand centre of volition, whereby the motor impulse to the peripheral nerves is arrested. Under the same heading, also, we have treated of those palsies due to general derangement of the cerebral circulation and nutrition, in which, the entire mental function having become arrested, no impression is made, and no voluntary motion can take place. Besides this, in a previous chapter, we have already considered the subject of paralysis arising from destruction of the fibres of the spinal marrow, through which the impulse from the seat of volition is conveyed to the motor nerves. The present section is exclusively devoted to the variety of palsy arising either from separation of the peripheral nerves from the brain or spinal marrow, or from a loss of irritability on the part of these nerves owing to alteration in their structure.

Etiology.—Separation of motor nerves from the central organs is not unfrequently the result of injury, and in this class belong the cases of section of nerves by a surgical operation, and by wounds of other kinds. In other instances the disturbance of continuity depends upon the extension of an ulceration or other destructive process to a neighboring nerve. The destruction of the facial nerve, during its cours-

through the canal of Fallopius in caries of the petrous bone, may be regarded as a prototype of this variety. A peripheral end of a nerve may also be separated from its central organ by the continued pressure of a tumor, an aneurism, or an exostosis which gradually consumes it. Sometimes even a temporary pressure upon a nerve has the same effect, which is probably owing to the continuity of the nerve's having become broken at the compressed point. Thus *Hasse* has seen a motor palsy of the arm which proved refractory to all treatment, in a person who had slept some time with his arm resting over the back of a chair. In very rare cases the disturbance of continuity of the nerve is due to a primary partial neuritis.

The structural changes whereby the motor nerves lose their excitability, without undergoing solution of continuity, are as unknown as are those which destroy the function of the sensory nerves. Thus cutting off the supply of arterial blood will arrest the irritability of the motor nerves without producing in them any structural alteration. It is probable, although not proved, that rheumatic palsy is due to hyperæmia and œdema of the neurilemma, causing compression of the nerve-fibres. Physiological experiment warrants the supposition that the paralysis, which sometimes arises after violent attacks of cramp, is the result of over-exertion; but a nerve which has lost its excitability through undue exertion does not differ appreciably from an excitable nerve. It is the same in case of a paralysis arising in consequence of a moderate pressure or strain. Here, from its favorable course, we must attribute the palsy to a diminution of the irritability of the nerve, and not to its destruction. Finally, the structural changes of the nerves are unknown, which give rise to the paralysis of lead-poisoning and of miasmatic poisoning, as well as in the so-called essentia palsies. (See Chapter XXI.)

Although hysterical palsy has not been counted either among the cerebral diseases or among those of the spinal marrow, it probably does not belong to the class of peripheral paralyses, but rather depends upon some impalpable anatomical abnormity of the centre of volition, as we shall explain more in detail when treating of hysteria.

Symptoms and Course.—Complete separation of a nerve from its central organ renders the muscles which it supplies incapable of motion, producing complete palsy or paralysis. Textural changes, which absolutely destroy the irritability of the nerve, have the same effect. When the nervous irritability is merely diminished, but not annihilated, feeble contractions still remain possible. This condition is called incomplete palsy or *paresis*.

A paralysis or paresis of peripheral origin is usually distinguishable from a central palsy: 1. By its extent. As we have seen, the charac-

teristic forms of cerebral and spinal palsy are hemiplegia and paraplegia, while a palsy limited to the range of influence of some particular nerve is an almost pathognomonic symptom of peripheral paralysis. It is only as a symptom of incipient sclerosis, and in rare instances, that the power of volition is suspended in single nerves owing to disease of the brain, or that the connection between the spinal marrow and any particular nerve is broken. The converse of this proposition is not true, however, as there are many peripheral palsies which involve large numbers of nerves. Even in such cases, however, the mode of extension of the malady often reveals its source. When a tumor of the brain causes palsy of the region supplied by the various cerebral nerves, the palsy does not arise in all of the nerves simultaneously, but extends gradually from one to another as the tumor grows. I shall mention one case observed in the Greifswalder clinic, as a striking example of how extension of the palsy may be of great importance as a means of distinction between spinal and peripheral paralysis due to caries of the vertebræ. The patient suffered from caries of the vertebræ, and his upper extremities were in a state of almost complete paralysis with anæsthesia, while the mobility and sensation of the lower limbs was quite normal. From the manner in which this palsy had developed, it could be decidedly inferred that it was the nervous trunks of the brachial plexus which were affected by the vertebral disease, and not the spinal marrow. Rheumatic palsy, which likewise not unfrequently affects a large number of nerves, does not present any peculiarity with regard to its manner of extension, and for a differential diagnosis we can only avail ourselves of the fact that it is not apt to assume the form of hemiplegia or paraplegia. On the other hand, the mode of development of lead-palsy is very characteristic, so that from it alone the real nature of the disease may be determined, and other forms of peripheral palsy, as well as the central palsies, may be excluded from the diagnosis. Lead-poisoning always affects the upper extremities first, attacking the extensors of the fingers, hands, and arms in succession, while the flexors remain quite free from disease.

2. If, from the outset of an attack of palsy, there have never been any derangements of the cerebral function, the origin of the disease is, in all probability, peripheral. The converse of this proposition also does not hold good; for a paralysis may be combined with serious cerebral disorder, and the paralysis may still be a peripheral one. Ample proof of this condition is given in cases of tumors at the base of the brain.

3. When the affected nerve is a nerve of mixed function, the complication of palsy with anæsthesia of the region known to be supplied

by its sensory fibres, is an important criterion of the peripheral origin of the disease.

4. The absence of reflex and sympathetic movements in the region of the palsied nerve is of similar import. If the continuance of the normal sensibility informs us that the function of the sensory nerves of a part is still preserved, and that its connection with the brain remains intact; and if, nevertheless, no reflex signs arise when the sensory nerves are irritated, there can remain no doubt that an interruption exists in the motor nerves, and hence that the palsy is peripheral. For instance, if a patient be unable to close his eye at will, and do not even wink when the bulb is touched, although the sensibility of the conjunctiva be normal, the palsy is certainly peripheral. The reverse of this condition is a still better proof of the central origin of a palsy. If a patient be unable voluntarily to bring a certain nerve into excitement, and, hence, unable to contract the muscles supplied by that nerve (for instance, if he cannot close the eye when told to do so; and, on the other hand, if the nerve be excited, and all its muscles made to contract if a sensory nerve be irritated, as when we touch the conjunctiva with the finger), we have to do with a central palsy. In such a case the irritability of the motor and sensory fibres is perfect, as is also the path by which the impression is conveyed from the centripetal to the centrifugal nerves; but the centre of volition in the brain, or the channel through which the influence of the will is imparted to motor nerves, is destroyed.

5. Finally, the early extinction of electric contractility in a nerve—that is, the absence of contractility of its muscles upon application of the induced current—is an important sign that the paralysis is of peripheral origin. The induction apparatus, the exaggerated and indiscriminate employment of which, for therapeutic purposes, should be discouraged, deserves a much greater employment as a means of diagnosis, especially in private practice, than it has obtained hitherto.

In recent cases of peripheral palsy, which come under our notice tolerably often in private practice, and where, owing to the greater prospect of successful treatment, it is of peculiar importance to know whether the disease be of peripheral or central origin, examination with the induced current will decide the matter almost with certainty. In many cases of peripheral palsy, the electric contractility of the muscles sinks to a minimum, even within a few days after the occurrence of the palsy, and soon afterward ceases entirely. In cerebral paralysis it is quite otherwise. There the electric contractility often continues unimpaired for months. Hemiplegic patients, indeed, are apt to be much impressed when the faradizer, by application of the electrodes, readily causes contraction of muscles, which for months have been be-

yond the power of their will, and they spare neither time nor money in order to subject themselves to a treatment in which—ineffectual though it be—they place the utmost confidence. The degeneration of peripheral nerves, which soon follows upon their separation from the central organs, and which we are able to demonstrate anatomically, accounts, in some measure, for the early extinction of electric contractility in peripheral palsy. Nor, for the same reason, should we wonder that it soon ceases in rheumatic palsy, and in cases of lead-poisoning. No matter how slight the disease from which the nerve suffers in such a case, it will always suffice to bring about in it such a change that neither the will nor the application of the induced current is capable of producing excitement in it. In cerebral palsy, also, a degeneration of the peripheral nerve finally sets in, as well as an atrophy and fatty degeneration of the unemployed muscles. Hence, in very old cases, loss of electric contractility can no longer be made use of as a means of distinction between central and peripheral palsy. In spinal palsy the electric contractility is sometimes long retained, while at other times it speedily ceases. Hence, it is of little worth in a diagnostic point of view. I believe that these differences may be accounted for, in some degree, from the results of the investigations of the structure of the spinal marrow by *Schroeder van der Kolk*. Where centrifugal fibres exist in the spinal marrow, which, instead of passing to the peripheral nerves, first go to the ganglion-cells from which the peripheral nerves spring, it would seem (arguing from analogy) that, upon destruction of this first set of fibres, a similar condition, as regards the electric contractility, exists, as obtains in cerebral palsy, while, upon destruction of the fibres proceeding from the ganglion-cells, the condition is analogous to that of a peripheral paralysis. It is remarkable that, in many cases of peripheral palsy, the muscles can be made to contract, by means of the constant current, while the induced current will fail of effect, and, still more strange, that streams so feeble as to excite no contraction upon the unaffected side will induce it upon the paralyzed side. We have no satisfactory explanation of this circumstance, which I have met with in two cases of rheumatic palsy in the course of a single semester. One thing, however, may be deduced from our previous remarks: that the character of the irritation produced in a nerve by the constant current, with its catalytic action, is essentially different from that caused by the induced current.

Separation of a motor nerve from the central organs and the extinction of its irritability induce the same derangements of circulation and of nutrition which we have described while treating of the peripheral anæsthesias, and such derangements are particularly severe in cases where anæsthesia and acinesis exist together. Lowering of the

temperature in paralyzed regions depends upon a retardation of the circulation. The quicker new blood reaches the peripheral region, which is constantly giving off heat, so much the less readily does it become cooled. On the other hand, the slower new warm blood enters the part, so much the sooner will its temperature assimilate itself to that of the surrounding region. Retardation of the circulation seems to me to be dependent upon a contraction of the arteries; at all events, the pulse is often smaller upon the palsied side than upon the sound one. We are as yet unable to account for this narrowing of the artery upon the paralyzed side. When a palsied part becomes inflamed, an elevation of its temperature is observed in it, instead of a depression.

We do not attempt to decide whether the incomplete anæsthesia, which usually develops by degrees in palsied parts (even where originally the disease has been a motor paralysis), depends upon retardation of the circulation, or whether it is to be ascribed to a transmission of the passive condition, through the ganglion-cells, from the motor to the sensory fibres.

The course of peripheral palsy exhibits great variety, according to the cause which produces it. Where a nerve has been cut through, the interrupted connection not unfrequently becomes restored, and the palsy gradually and completely disappears. If, however, a large portion of a nerve be destroyed, the palsy remains stationary throughout life. The progress of rheumatic palsy usually is favorable, and it generally terminates in complete recovery. This is also the case with the exhaustion of irritability resulting from convulsions, and from the incomplete traumatic palsy caused by slight injuries. The paralysis which sometimes remains after recovery from typhus also admits of favorable prognosis, while that arising from lead-poisoning is exceedingly intractable, and often proves incurable.

Treatment.—The fulfilment of the causal indication in peripheral palsy is but rarely possible, and even then is seldom followed by beneficial results. Thus the extirpation of a tumor, which has caused paralysis by pressure upon a nerve, scarcely ever restores normal motion of the part.

Rheumatic paralysis forms an exception to this rule, when not of too long standing, as a proper treatment of the primitive disease in such cases often furnishes the best results. However, this is not to be anticipated from the use of those very uncertain remedies, colchicum, quina, and aconite, but rather from the methodical employment of warm baths. Every year troops of paralytic patients, curable and incurable, crowd to Teplitz, Wiesbaden, Wildbad, or Pfäffers. The reputation which these watering-places enjoy, especially in the treatment of palsy, is mainly due to their really surprising efficacy in rheu

matic paralysis. As corroborants of the bath-treatment, but only in cases where, owing to unfavorable circumstances, bathing is impracticable, we recommend the application of stimulants to the skin, applied along the course of the affected nerve. The usual prescription of an embrocation of aromatic tinctures, which irritate the nose more than the skin, should be set aside in favor of rubefacients and issues.

Of the remedies called for by the indication from the disease itself, the application of the constant electric current is that which deserves our greatest reliance. We have already expressed our opinion that cures effected by the constant current probably depended solely upon its catalytic action, and that, by means of the galvanic treatment, it is in our power greatly to modify the circulation, the process of endosmosis, and nutrition itself, even in tissues lying deep beneath the skin. The induced current does not have this effect, and we have seen many cases of peripheral palsy, which had been treated unsuccessfully by the induced current, healed when the constant stream was applied. This is especially the case with the rheumatic and traumatic palsies, and those induced by lead and other poisons.

The symptomatic indication demands: 1. That we should preserve what remains of irritability in a partially paralyzed nerve, and avert, if possible, its complete extinction. 2. That we should prevent atrophy and fatty degeneration of the palsied muscles, or check them where they have already begun. Both complete extinction of the already reduced irritability, and the atrophy and degeneration of the palsied muscles, are mainly due to continued rest, and to lack of excitement, which thus constitute a new factor, whereby a palsy, dependent upon other causes, is rendered more severe and intractable. It may even furnish the sole reason why a paralysis merely improves sometimes, without disappearing entirely. For the prevention of such a mishap, localized faradization is an invaluable remedy. It is an important rule, in its employment, not to protract the sittings too much, and not to employ too strong a current. Since we know that the excitability of a nerve is quite as liable to impairment or destruction from too much exertion as from too much rest, this rule needs no comment. It is equally evident that, in traumatic, rheumatic, and toxic palsy, the induced current is not to be employed before the disturbance of continuity has been allayed, and the excitability of the affected nerve has begun to return; or, in other words, that the current is not to be applied until we find that the muscles begin to contract under its influence. It is very desirable that every physician should make himself so familiar with faradization localizée that he need not leave its employment to some one else. The labors of *Ziemssen* have rendered this task quite an easy one. A few days of practice upon the healthy subject, under the in-

structive guidance of this book, suffices to impart the degree of skill requisite to impress the uninitiated.

The curative effect of strychnine upon peripheral palsy may be placed by the side of that of the induced current. Nor does strychnine at all tend to promote the union of divided nerves, nor repair the structural changes which have caused the palsy. On the other hand, it is probable that this drug, by exciting reflex action in the spinal column, and through the augmented reflex excitement thereby induced in the motor nerves, may stimulate the irritability of the latter, where it is not already completely extinguished. In order to obtain results from strychnine, doses must be given of sufficient size, and must be kept up long enough to produce visible effect upon the reflex action of the spinal marrow—that is, until slight twitching is induced. We prescribe either the alcoholic extract of nux vomica, one-third of a grain, gradually increasing up to two grains, or the nitrate of strychnine, in dose from the twelfth to the fourth of a grain. Other medicaments, such as arnica and rhus toxicodendri, scarcely have any effect upon peripheral palsy.

CHAPTER XX.

PALSY OF THE FACIAL NERVE—MIMIC FACIAL PALSY—BELL'S PALSY.

Etiology.—In the coming chapter we leave unnoticed the variety of facial palsy arising from suspended volition, which is almost always accompanied by hemiplegia, and constitutes a common symptom of apoplexy, and of other diseases of the brain.

The irritability of the facial, or of its attachment to the brain, may be impaired: 1. By causes which act upon it prior to its entrance into the internal auditory meatus. 2. By such as affect it during its course through the petrous bone. 3. By agents which involve the peripheral ramifications upon the face. Within the cranium the facial nerve is most frequently compressed or destroyed by cerebral tumors springing from the base of the skull, or which have advanced toward its base. More rarely it proceeds from exudation, from thickening of the dura mater, or from exostoses. In the canal of Fallopius the nerve is more frequently destroyed by caries of the petrous bone, although one or two cases have been observed in which fractures and gunshot wounds have caused injury of the facial within the petrous bone. The peripheral branches are sometimes cut, either by accident or intentionally, during surgical operations. Thus it was with the coachman who was so thankful to *Bell* for the successful extirpation of a tumor about his ear, but who complained that since the operation he could no longer whistle to his horses. The continuous pressure which the ramifications

sometimes are subjected to by enlarged lymphatic glands, or other tumors, as well as contusions and concussions, resulting from blows upon the ear, may give rise to facial palsy. It is not unfrequently observed in newly-born children, when branches of the nerve have been bruised by the forceps during labor. Sudden chilling of a warm face, however, is a much more common source of facial palsy than any other agent. Many patients acquire it by looking out of the window immediately after rising in the morning. *Halla* attributes the increasing frequence of facial paralysis to the railroads. People hurry to the station, arrive there warm, enter the carriage, and expose their face to the draught of the window. The result is a palsy of the face.

The affection is a somewhat common one, so that *J. Frank* has seen twenty-two cases of it in the course of fifteen years. Statistics as to its frequence at various ages, and in different sexes, and different sides of the face, have not led to any important conclusions. If it be true that the affection is more common upon the left side of the face, it is not on account of any predisposition of the left facial nerve, but because of the greater exposure of the left cheek to blows on the ear, and to other injuries.

Symptoms and Course.—The symptoms of facial palsy consist in an immobility and relaxation of the facial muscles, which are supplied by the affected nerve-fibres. Palsy of the frontal muscle, and of the corrugator supercilii, makes it impossible to wrinkle the forehead. *Romberg* says: "The old man's forehead becomes as smooth as that of a child, and there is no better cosmetic for old women." Palsy of the orbicularis palpebrarum prevents the patients from shutting the eye completely. If told to do so, they lower the lid a little, by relaxing the levator palpebræ superioris, over which they still have control, and roll the bulb upward, so as to hide the cornea. The tears are no longer conducted to the lachrymal puncta, but flow down over the cheek. The eye, being imperfectly closed, and exposed to all manner of injuries, readily inflames. The levator labii superioris, alæque nasi, the levator anguli oris, and the zygomaticus, are unable to draw up the upper lip, nose, or commissure of the mouth, and to dilate the nostril. The buccinator being disabled, the cheek puffs out during expiration, like a loose sail. Some of the purposes for which the facial muscles are employed, the pronunciation of the labial letters, whistling, blowing, and expectorating, now fail. Even in chewing, which process, being independent of the facial, goes on undisturbed, the morsel, when on the affected side of the mouth, often falls between the teeth and the cheek, and has to be disengaged thence by the fingers. When the paralysis involves the whole of one side, there is a remarkable distortion of the countenance with every play of expression. This arises

partly from the fact that muscular contractions only occur on the sound side, while the other remains motionless, and partly because the contracting muscles upon the sound half of the face are not counterbalanced by the palsied half, so that the countenance is drawn to one side. Even during rest the face remains more or less distorted and unsymmetrical. The palsied angle of the mouth is lower than the healthy one, the nostril is narrower, and all the pits and depressions are effaced. The point of the nose and the mouth are drawn over toward the sound half of the face. All this deformity is due to the want of balance between the healthy muscles of one half the face and their palsied antagonists. In the same way there is lagophthalmos of the affected side, from a preponderance of the unpalsied levator palpebræ superioris over its palsied antagonist, the orbicularis palpebrarum. When paralysis of the facial is bilateral, the face becomes void of all expression, and the patient laughs and weeps without exhibiting any play of countenance. I have never seen such a case myself, but can easily believe that the aspect of a person whose face remains motionless, even while he is laughing loudly, will present the hideous appearance of a mask. The impairment of the sense of taste, the distortion of the uvula, and deviation of the tongue, though less obvious symptoms, are equally constant. It is uncertain whether the obtuseness of the sense of taste depends upon diminution of the secretion of saliva, and consequent dryness of the mouth, or whether the chorda tympani nerve, by erecting the papilla of the tongue, aids the gustatory sense. Displacement of the uvula to the sound side is accounted for by the fact that the motor nerve-fibres, passing through the nervus petrosus superficialis major to the sphenopalatine ganglion, from which the descending palatine branches proceed, only contract the muscles of the uvula upon the side which is not palsied. This explanation, however, is not absolutely satisfactory.[2]

Regarding the deviation of the tongue, we refer to what we have already said while treating of apoplexy, as this symptom is of more common occurrence in cerebral than in peripheral paralysis. In a previous chapter we have given a detailed explanation of why the reflex motion should be impossible in complete peripheral palsy of the facial, and why the electric contractility of the muscles of the face soon becomes extinct. In recent and uncomplicated cases, the sensibility of the paralyzed half of the face is normal. When of longer standing, it generally becomes somewhat blunted, probably in consequence of nutritive disorder of the region supplied by the affected nerve. This shows itself chiefly through emaciation and flabbiness of the palsied part of the face, by the disappearance of the fat, and by the shrivelled condition of the skin.

According to *Romberg*, the following are the signs whereby we may recognize whether the source of the paralysis is to be sought within the cranium, in the petrous bone, or upon the face itself: 1. We may infer that the affection originates in disease at the base of the skull, when there is participation of other cerebral nerves in the palsy, as shown by squinting, deafness, anæsthesia, etc., as well as by the simultaneous occurrence of palsy of the extremities of the other side of the body. 2. Evidence that the palsy originates in a disease or destruction of the facial nerve within the canal of Fallopius consists in long-standing otorrhœa, hardness of hearing or complete deafness, obliquity of the uvula, dryness of the mouth, perversion of the sense of taste, which depends upon implication of the nervus petrosus superficialis minor and chorda tympani, which are never affected except in this form of the disease. 3. We know that the palsy proceeds from the peripheral ramifications, when the disease is plainly dependent upon exposure to cold, or upon some violence done to the face, or upon the pressure of a tumor upon the facial nerve, especially in the region of the stylo-mastoid foramen, the ear itself meantime being sound, the taste normal, and the uvula straight.

The course of facial palsy varies greatly, according to the seat and nature of its cause. Where the nerve has been destroyed by a tumor at the base of the skull, or by caries of the petrous bone, the palsy is, of course, incurable, as is also the case when a tumor has broken the continuity of the nerve by continued pressure upon its peripheral branches. When the affection proceeds from exposure to cold, or from slight injuries, the prognosis is better. This is especially true of the congenital palsy induced by pressure of the obstetric forceps. But, even in adults, it by no means invariably happens that traumatic or rheumatic paralysis disappears in the course of a few weeks or months.

Treatment.—From what has been stated regarding the prognosis of facial palsy, the rheumatic and traumatic forms of the complaint are the only ones susceptible of treatment. Although they usually recover without interference, it is better, in recent cases of traumatic origin, to employ local antiphlogistics. Whenever an adult has a palsy of one side of the face, caused by a blow or shock, we should prescribe leeches and cold compresses, and should rub the part with mercurial ointment. Congenital paralysis, also, when of traumatic origin, may be left alone. When the disease arises from cold, and is recent, the affected side of the face is to be covered with well-wrung cold compresses, covered with oil-silk or india-rubber cloth, which are not to be changed until after the lapse of several hours. The face may also be enveloped in cotton wool, and a vapor-bath may be taken. At a later period stronger irritants may be used: lotions of essence of

mustard, mustard-plasters, embrocations of croton-oil, blisters, etc. A great number of cases are on record in which rheumatic facial palsy has been cured by the application of the constant galvanic current. In two cases, I myself have succeeded in effecting a cure, although but a gradual one, by employment of galvanism. In each of these cases, treatment by the induced current remained without effect. Where the electric contractility can be reëstablished by localized faradization, the cure will be promoted and hastened by the methodical use of the induced current.

CHAPTER XXI.

PALSY OF THE SERRATUS MUSCLE.

Etiology.—In rare instances, two of which have come under my personal observation, palsy of the serratus has formed part of a myopathic paralysis, involving many of the muscles, the result of the so-called progressive muscular atrophy (see appropriate chapter). The affection is much more commonly confined to the serratus alone. This fact, and the circumstance that the long thoracic nerve is not distributed to any other muscle except to the serratus anticus magnus, would imply that most cases of palsy of the serratus are neither of cerebral nor of myopathic origin. We have laid it down as an important sign of the peripheral nature of a palsy, that the affection involves the region supplied by some particular nerve, and while neighboring muscles supplied by other nerves remain sound. Besides this is the well-known fact that peripheral palsy and peripheral neuroses by preference affect the region supplied by nerves which pass through long, narrow channels and holes, like the long thoracic, which runs through the scalenus medius. This very important point in the pathogeny of palsy of the serratus, and the proof of the fact that, in most of such cases, we have to deal with a peripheral neuropathic affection, have not hitherto obtained the notice which they deserve.

The exciting causes of palsy of the serratus are usually obscure. It is plain that the long thoracic nerve, in its passage through the scalenus medius, may suffer injuries which escape our attention. In some instances, over-exertion of the upper extremities, or cold, has been the assigned cause. In one observation, reported by *Neuschler*, the palsy of the serratus was preceded by a fall, and a tumor afterward formed in the neck, which the physician in charge proposed to open by incision. In one very interesting case, which came under my own observation, a carpenter, who used to carry the heavy beams for buildings upon his right shoulder, had to employ the left shoulder instead,

on account of the development of a palsy of the right serratus muscle. Some time afterward, palsy of the left serratus also began to appear.

Symptoms and Course.—Palsy of the serratus muscle is easy of recognition, as the absence of its function and the undue action of its antagonistic muscles occasion characteristic deformities and disorders of motion. The function of the serratus is to press the scapula against the thoracic wall, and to draw its lower angle downward and outward. The serratus is especially required in the act of elevating the arms above a horizontal line, as it then draws the lower angle of the scapula outward, and turns the glenoid cavity of the joint upward. It is by this act alone, and not by the contraction of the deltoid, that we are enabled to lift the arm above the shoulder. When the serratus is paralyzed, the inner border of the scapula, and particularly its lower angle, instead of lying against the chest, stands up like a wing, drawing up a three-cornered fold of skin before it, and admitting of our reaching deeply into the subscapular fossa. At the same time, the lower edge of the scapula stands up too high, and too far inward. The antagonistic muscles, the trapezius and the levator scapulæ, have drawn the superior angle upward, and the weight of the arm and the pectoralis minor have depressed the external angle, and thrown it forward. The patient is unable to lift his arm above a horizontal line, and is thus rendered extremely awkward. Any one, who has often watched a patient with this affection put on or take off his coat or shirt, will be able to make a diagnosis in the next case he meets with, from these acts alone. If we press the inferior angle of the scapula against the chest, and, at the same time, push it in, the patient is once more enabled to lift his arm above his shoulder without difficulty. Palsy of the serratus is an obstinate complaint. In none of the cases which I have seen was a complete cure effected. In many instances, however, the patients so far improved as to be able to do easy work.

Treatment.—The most commendable remedy, in recent palsy of the serratus, is local blood-letting and derivation to the skin, applied over the point where the long thoracic nerve passes through the scalenus medius. In chronic cases, according to our present experience, faradization does not promise much benefit. On the other hand, the constant current is strongly to be recommended. Unfortunately, however, hitherto I have had but one opportunity of applying it. The cures obtained by means of the constant current, in cases of peripheral facial palsy, which I consider to be quite analogous with palsy of the serratus, urgently demand the treatment of the latter by galvanism.

CHAPTER XXII.

PROGRESSIVE PALSY OF THE CEREBRAL NERVES—PROGRESSIVE PALSY OF THE TONGUE, VELUM PALATI, AND LIPS—GLOSSOLARYNGEAL PALSY.

A SERIES of observations have recently been made, particularly by French writers, of a paralysis, which, commencing at the lips, extends successively to the tongue, palate, pharynx, and sometimes to the muscles of the glottis, more rarely to those of the eyes. In all cases hitherto observed no coexistent derangement has been perceptible in the sensory nerves, nor in those of special sense.

The pathogeny of this peculiar disease is obscure. The only lesion constantly found *post mortem* is an intense fatty atrophy of the palsied nerves, especially the hypoglossal nerve. In some cases a diffuse sclerosis of the medulla oblongata has also been found. According to an hypothesis which is as yet unproved, advanced by *Wachsmuth*, who proposes to call the malady in question "bulbar palsy," the disease consists in a "central affection of the medulla oblongata, having its seat in the olivary bodies, and in the gray matter lying far back between the diverging lateral and posterior columns, and which forms the floor of the fourth ventricle. According to *Wachsmuth*, the atrophy of the nerves is secondary, and a consequence of the degeneration suffered by the nuclei at the points above named. In some cases the palsy of the region supplied by the cerebral nerves was preceded by the symptoms of an incipient, progressive muscular atrophy, and the majority of French authorities regard the two diseases as closely related, although, in the progressive palsy of the cerebral nerves, no diminution in size is observable in the affected muscles. At the outset the patient makes but little complaint. He cannot pucker up his mouth, and hence cannot blow, whistle, nor spit. The saliva which collects in the mouth runs from it involuntarily. The expression of the countenance becomes blank and strange, as the muscles of the lips cannot take part in the play of expression. Pronunciation of the labial letters is difficult, and gradually becomes impossible. If the malady extend to the tongue, not only does articulation become still more embarrassed, but the acts of chewing and swallowing become impeded; and afterward, when the tongue has become still more helpless—lying quite motionless in the mouth—these acts are no longer practicable. Palsy of the palate shows itself from the nasal tone of the voice, and, as long as the pharyngeal muscles remain sound, by the regurgitation, through the nose and mouth, of food and liquids which enter the pharynx. If the pharynx also become palsied, the patient,

when offered to drink, rejects a large portion of the liquid with violent expiratory motions, so that at last it often becomes necessary to feed him by means of the œsophagus tube. The choking-fits thus provoked by attempting to drink, and the rejection of the liquid amid spasmodic coughing, might awaken the suspicion that a communication exists between the larynx and pharynx, or œsophagus. In one case of this kind, my colleague *Bruns* demonstrated, by means of the laryngoscope, that, "if the patient merely was fed with small spoonfuls of milk or porridge, the swallowed portion only passed as far as the pouch which lies behind the larynx. If, by further swallowing, the level of the liquid rises so as to pass the bottom of the notch between the arytenoid cartilages, the so-called rima glottidis posterior, the soft liquid mass immediately flows forward through this opening into the larynx, and instantly gives rise to a fit of coughing."

This disease is generally a tedious one. Death is the usual result, either from impairment of the general nutrition of the patient, owing to the difficulty with which he obtains nourishment, or else from violent bronchitis, or pneumonia, in consequence of the repeated intrusion of liquid into the air-passages. Hitherto no authentic case of improvement or cure has been reported. *Benedikt* is the only one who claims that, by galvanization of the sympathetic, and at the mastoid process, he has obtained important success and even a cure, and in advanced cases has relieved dangerous symptoms, such as difficulty in swallowing. For this purpose he applied the copper-pole to the spinal column, and strokes with the zinc-pole upon the pomum adami and neighboring parts, so that in each sitting he is able to induce some twenty or thirty acts of deglutition. May there not have been some confusion with hysterical dysphagia in these almost miraculous cures? I have cured a hysterical patient by a treatment almost purely psychical, who for months previously had been fed through a tube, and had carried a canula in her trachea for an equally long period of time.

CHAPTER XXIII.

ESSENTIAL PALSY OF CHILDREN—SPINAL INFANTILE PALSY (*Heine*).

ETIOLOGY.—Whether the essential palsy of children be a disease of the brain, of the spinal marrow, or of the peripheral nerves, or whether it proceed from all or any of them in turn, as *Vogt* supposes, is a matter which, in our present ignorance of its *post mortem* lesions, cannot be positively decided. The name "essential palsy" I hold to be a most appropriate one. Although the paralysis originally may have been due to an inflammation or effusion in the spinal marrow,

yet, at the time when the palsy is called an essential palsy, all such processes have long since subsided, and it is not the primitive disease, but the paralysis and its consequences, that we have to treat. Essential palsy as a rule is the permanent product of a very acute process; for all observers agree that the affection develops within a few hours, and that it never extends from the limb first attacked to the other limbs.

The causes of the disease are as obscure as is its pathogeny. It occurs almost exclusively among children during the period of dentition, and for a short time afterward; that is, from the sixth month to the third year of life. Girls and boys are equally liable, as are also scrofulous and cachectic subjects, and those previously robust. The assigned causes have been the acute exanthemata, and cold, especially allowing children to sit upon cold stones. The latter idea is unworthy of attention, as innumerable children sit daily upon the stones, while essential palsy is not at all a common disease.

Symptoms and Course.—In many instances the attack commences with febrile symptoms, and signs of cerebral hyperæmia or meningitis. Mental excitement, convulsions, loss of consciousness, gnashing of the teeth, are common to this, as well as to the other two maladies above mentioned; and as in such cases it often happens that it is only by the course of the disease that we can decide whether we have a meningitis or a cerebral hyperæmia to deal with, so here, too, we have no means of distinguishing the primary stage of an essential paralysis from that of hyperæmia of the brain, save that, after subsidence of the convulsions and return of consciousness, a total paralysis of one or more extremities remains. Sometimes one foot, sometimes one hand is affected, sometimes both lower extremities; but it never happens that both extremities of the same half of the body are paralyzed, a fact which very plainly implies the independence of the disease from cerebral apoplexy or encephalitis. The bladder and rectum never take part in the palsy. A mode of commencement of the malady, which is scarcely less common than that by violent cerebral symptoms, consists in an undefined attack of feverish symptoms, during which, without precursory convulsions or stupor, an arm, or leg, or both legs, suddenly hang useless, and are entirely incapable of voluntary motion.

The subsequent course of the disease may vary. Sometimes the palsy disappears in a day or two, and the disease ends in complete recovery. *Heine*, who indisputably has seen the largest number of cases, doubts whether such examples (to which the name "temporary infantile palsy" has been given) proceed from the same causes which give rise to the stationary, permanent essential paralysis. *Duchenne* noticed that, in recent cases of the temporary form, the electric con-

tractility of the muscles was retained; whereas, in recent cases of the permanent form, a few only of the muscles of the paralyzed limb maintained their electric contractility, while in the others it was lost. These observations may be summed up as follows: that the prognosis of an essential palsy is favorable, and that a cure is to be hoped for, when the affected muscles preserve their contractility; but that it is unfavorable, and that degeneration and atrophy ensue, when the electric contractility is extinct.

When the disease passes into the so-called second stage, the relaxed flexible soft limbs, which may readily be placed in any required position, gradually lose their original plumpness. They suffer an atrophy which involves the skin, fat, muscles, and even the bones. In the course of a year the circumference of the limb, and even its length, is far smaller upon the crippled than upon the sound side. The pulse is small, corresponding to the deficient nutritive condition and marked depression of temperature of the paralyzed limb. It has a livid hue, and is liable to bed-sores, chilblains, and ulceration. As the disease progresses, deformity and contractions of the crippled limb are added to the atrophy. The wasted deltoid muscle is often no longer able to hold the head of the arm-bone in its socket on the shoulder-blade; so that the arm sinks, stretching the capsule of the joint by its weight. We then find a depression immediately beneath the acromion, the head of the bone lying farther downward and backward. It is easy to replace the bone; but, as soon as the arm is permitted to hang down, it is immediately redislocated by its own weight. When the lower extremities are attacked, particularly if it be at the period when children try to move themselves about by shuffling, permanent shortening takes place in the muscles which still retain some degree of contractile power and encounter no resistance from their antagonists. This is the mode of origin of a variety of forms of clubfoot, flexions of the hip, and the deformities of the knee-joint known as genu valgus. In advanced periods of the disease, the electrical contractility of the degenerated and wasted muscles is extinct. No conclusion as to the origin of the affection can be drawn from this condition, as it occurs in all forms of paralysis (even in the cerebral form) after it has lasted long enough for the nerves and muscles to degenerate.

The general health usually remains unimpaired in essential paralysis. Many patients attain a great age, and, when they belong to the lower classes of society, are often seen as mendicant cripples on the high-roads.

Treatment.—It is only in very recent cases of essential palsy that any benefit is to be expected from local blood-letting, or from derivatives by the side of the spine. In old cases we have as little reason

to expect the resolution of the remains of a process which has long since subsided, as to restore the apoplectic cicatrix of the brain after an attack of apoplexy, and thus to cure the hemiplegia. Indeed, where the electric contractility is extinct, and the nerves and muscles have degenerated, the restitution of the spinal marrow to a state of health would be of no benefit to the palsy.

Although we scarcely ever are able to fulfil the causal indications or the indications from the disease, yet a treatment of the symptoms of the disease has been followed by comparative success. As long as any of the muscles maintain the slightest trace of their contractility, the systematic and active application of the induced current is indicated as being the best and surest means of preserving and increasing what remains of the irritability, and of arresting the atrophy and degeneration of the muscles. It is proved, moreover, by the results which *Heine* has obtained at his institution, that, even in cases apparently of the most desperate nature, the lot of the unfortunate patient may be materially alleviated by means of tenotomy and other expedients of rational orthopedic surgery.

ADDITIONS TO THE REVISED EDITION OF 1880.

SECTION III.—DISEASES OF THE PERIPHERAL NERVES.

1.—P. 331.

Neuralgia is occasionally so obstinate that, besides exhausting the patient, it exhausts the physician's list of remedies ; and it may not be out of place to record some of the numerous modes of treatment recommended.

Apply ice to the seat of pain as long as the patient can bear it, while he holds brandy in the mouth.

In periodic cases, where quinine fails, try salicylic acid in 30-gr. doses repeated two or three times ; or hypodermic injections of Fowler's solution—arsenic ♏ v–x, with the same quantity of distilled water.

Granules of $\frac{1}{100}$ gr. of aconitia, repeated three times daily till they cause tingling, are said to give relief ; not to be used in cases of heart disease.

Inhalations of ether, of chloroform, or of nitrite of amyl (gtt. iij); if the latter is not effectual, it may be repeated in fifteen minutes and increased to six or eight drops ; it is more apt to give relief in cases where the face is pale.

Either the induced or constant currents of electricity may do good, though the latter is most reliable in neuralgia. Change of air; where there are spasmodic contractions of the muscles of the face, compression of the carotid on that side; and where there is paresis on the neuralgic side, compression of the opposite carotid, are said to do good.

Phosphorus gr. $\frac{1}{24}$, in mucilage, may be given ten minutes after something has been taken into the stomach, and repeated thus every two hours for three doses, then every three or four hours.

2.—P. 368.

Implication of the chorda tympani causes change in the sense of taste, especially for sour or salty things, on the anterior two-thirds of that side of the tongue. If it be true that the gustatory filaments of the facial only belong in the short portion between the entrance of the chorda tympani and the departure of the petrosus superficialis major, and pass with the latter to the second branch of the trifacial, loss of taste in facial paralysis would show that the cause must be at this part. Diminution of saliva has also been observed. Deafness may occur from disease of the internal ear or auditory nerve. There may be excessive sensitiveness of hearing (*hyperakusis*). In rheumatic paralysis this is said to occur especially for the deeper musical tones, and is probably due to paralysis of a small twig going to the stapedius muscle, and a consequent preponderance of the tensor tympani muscle.

Althaus says that in *rheumatic* paralysis the farado-muscular excitability is generally normal; there may be slight anæsthesia of the skin; the supinators and flexors are equally affected. In *traumatic* paralysis the farado-muscular contractility is lost; there is anæsthesia of the skin; the supinators and extensors are equally affected. In *lead* palsy the farado-muscular excitability is lost; there is no anæsthesia of the skin; the supinators are healthy. In *cerebral* paralysis the electro-muscular contractility continues. These diagnostic points are true in other paralyses as well as in that of the facial nerve.

SECTION IV.

GENERAL NEUROSES, OF UNKNOWN ANATOMICAL ORIGIN.

CHAPTER I.

CHOREA—CHOREA ST. VITI—ST. VITUS'S DANCE.

ETIOLOGY.—St. Vitus's dance may be called a purely motor neurosis, all of its symptoms being attributable to a morbid irritability of the motor nerves, while no derangement, or, at least, no constant derangement of the sensory or intellectual function can be detected.

The pathogeny of chorea is obscure. None of the anatomical researches hitherto made upon the subject, nor any study of its symptoms, give us any positive information as to the real point whence the morbid irritation of the motor nerves proceeds. The results of the somewhat rare autopsies which have been made upon subjects who have died of chorea have been either negative, or else so discordant that any lesion discovered in the central organs of the nervous system cannot be referred to the chorea, but rather to some accidental complication, or to the disease of which the patient died. The general implication of nearly all the cerebro-spinal motor nerves altogether contradicts the supposition that the origin of the disease lies in the peripheral nerves. The complete integrity of the other cerebral functions makes it improbable that the movements of chorea originate in the brain. On the other hand, certain pauses in the muscular restlessness which occur, particularly during sleep and during the action of chloroform, would seem to imply that the motor influence is derived from the brain rather than from the spinal marrow. There is no good ground for the hypothesis that chorea is dependent upon a disproportion in size between the spinal canal and the spinal marrow, or upon inflammation of the vertebræ, or upon spinal irritation, for we do not even know that the seat of the malady really lies in the spinal marrow.

In considering its etiology, in the first place, we are struck by the prevalence of the disease at the time of the second dentition and at the period of puberty. It often happens that an individual suffers from the disease at both these periods, and remains free from it during the interval. This accounts for the belief of the laity, that the complaint returns every seven years. Before the sixth year of life the disease is rare; and it is equally uncommon after the age of fifteen. Even the most advanced old age, however, is not entirely secure from it, and in such cases the affection exhibits a peculiar intractability. The predisposition to the disease is far greater in the female sex than in the male; and in certain cases an almost unmistakable hereditary tendency to St. Vitus's dance has been observed. Moreover, hydræmia, anæmia, and rheumatism seem to augment the tendency to this affection. It undoubtedly is going too far to regard the connection between chorea and rheumatism as constant; but it cannot be denied that a remarkable number of chorea patients have already suffered from acute or chronic rheumatism, or else, during the disease or after it has subsided, a rheumatic attack sets in. In like manner, inasmuch as false heart-murmurs are very often heard in the hearts of chorea patients (and although many of them may be considered as blood-murmurs, and due to anæmia and to nervous derangement), yet the number which are certainly dependent upon valvular disease is large enough to enable us to judge how many of the patients have already suffered from rheumatic pericarditis and endocarditis. One of the most severe cases of chorea which I have ever witnessed was in a girl fifteen years of age, each one of whose joints successively became swollen; and another was in a girl, twenty years old, who had bad disease of the heart. Besides the mimic instinct, various mental emotions, especially fear, have been assigned as causes of chorea, as have also the irritation of worms in the intestines, onanism, pregnancy, and other agents. In particular instances it of course is very difficult to determine the causal connection between chorea and such influences, which so often exist without perceptible effect upon the health. The effect of example, which plays the principal rôle in the chorea major as well as in chorea minor, is instanced in the epidemic appearance of the disease sometimes observed in boarding-schools. The influence of pregnancy is inferred from the fact that, among adult patients, very many of them are pregnant women. Chorea rarely appears before the end of the second month of pregnancy, and its appearance is equally rare in the later half of the term. Once established, it usually lasts until after delivery.

Symptoms and Course.—St. Vitus's dance is characterized by movements of the voluntary muscles, which, however, are not excited

by volition, but take place against the will of the patient, his consciousness meantime being perfectly unclouded. These movements go on not only at times when the patient does not intend to move, but also when he moves voluntarily. In the latter case, as the voluntary motion is complicated by the involuntary, the proposed action of the patient either is frustrated or carried out imperfectly or awkwardly. The involuntary movements of chorea are distinguished from the more simple and monotonous jerking muscular contractions of epileptic or hysterical attacks, by a great degree of variety, and by a sort of combination which imparts the aspect of design to the motions. A cursory and superficial observer would be much more apt to overlook and mistake the former than the latter.

In most instances the disease begins very gradually, and is not recognized for some time. It may be noticed, perhaps, that the sick child drops and breaks things a good deal; that it does not sit still; that it writes badly, or makes more mistakes than usual in playing on the piano, and it is accordingly scolded or punished that it may be more careful and correct its awkwardness. The poor child often does not know what it has done, and, in consequence of the unjust reproaches, becomes either depressed and sad, or else grows irritated and perverse. The restlessness of the muscles, meantime, becomes more and more apparent. The acts of awkwardness become more frequent, and are grosser than ever. The child misses in reaching for its tumbler, pricks itself with its fork, or makes extraordinary grimaces. The morbid character of this condition often becomes apparent to the minds of its relatives quite suddenly, and without the occurrence of any special change in the symptoms. It is much more unusual for the disease to develop suddenly, and from the outset to present the remarkable symptoms which characterize St. Vitus's dance in its later stages.

In pronounced chorea, the utmost variety of motions follow each other, in a manner so manifold and grotesque that the term "insanity of the muscles," which has been applied to it, seems quite appropriate. In the face, the eyebrows are alternately contracted and separated, the forehead wrinkled and smoothed, the eyelids rapidly winked, and now and then are fast closed for a moment. The eyes roll hither and thither, the mouth is successively pursed up, closed, then suddenly opened and shut, now spreading into a smile, now drawn down as if to weep, while the tongue is often and suddenly thrust forward. The head itself is turned, now forward, now backward, now sideways; the shoulders are raised and sunken. The upper extremities are flourished about. In the elbows, hands, and finger-joints, flexion and extension, pronation and supination, abduction and adduction, alternate with one another. Similar movements, which, however, usually are of less

active character, are observed in the lower extremities. The muscles of the trunk also take part in the general restlessness, so that the spinal column is inclined either forward, backward, or sideways; sometimes in one direction, sometimes in another. If the patient be lying in bed, he often is tossed upward, or toward the foot of the bed, or is even pitched out of it. In the worst forms of chorea, the patients are quite unable to sit upon a chair, but immediately slide off from it to the ground. This morbid restlessness grows all the more intense and general, if the patients pay attention to it, especially if they know that they are being watched. Sometimes it is stronger upon one side than upon the other, or is chiefly confined to one or more limbs. The muscles of the larynx and those of respiration but seldom take part in the disease, and the sphincters and the muscles of the pharynx probably never do so. Owing to the constant motion, it is difficult for the patients to go to sleep; when once asleep, however, the muscular twitching ceases. Occasional exceptions to these rules are now universally ascribed to dreams of movement, ever since *Marshall Hall* originally suggested the idea.

Nearly all the motions of the body, with exception of those of respiration and deglutition, are seriously embarrassed by this restlessness of the muscles. The articulation becomes indistinct, as the intentional movements of the mouth and tongue are accompanied by others which are unintentional. In eating, the fork misses the lips; in drinking, the beverage is spilt, so that a patient often has to be fed. Some of them have great difficulty in offering their hand, others are unable to dress and undress themselves. Even in slight cases all occupations requiring delicacy of manipulation become impossible, as, even in walking, the feet only touch the ground by a circuitous route, and, as the entire body is constantly making superfluous and irregular movements, there is something very remarkable and characteristic in the gait of the patient.

The other functions and the general health suffer comparatively little. It is easy to comprehend why the patient should be dispirited, sensitive, and wilful, when we consider that for weeks he has not been master of his motions, and that he has been constantly taunted about the mistakes he makes. Not unfrequently the grimaces of the patient are so at variance with his actual humor, or so unsuitable to the subject of conversation, as to give him a foolish, imbecile look, although his mind is quite sound. When of long duration, however, the accuracy of judgment seems really to be impaired, and other intellectual disorder arises. Sometimes, but not always, there is complaint made of headache and pain in the back. Although, strange to say, there is no especial fatigue of the muscles, the joints of the limbs are some-

times swollen and painful from the constant strain and motion. Unless there be some complication, there is no fever, although the pulse usually is accelerated. The appetite, digestion, secretion, and excretion do not present any constant peculiarity. When the disease has been of long duration, the nutrition of the patient suffers in consequence of the loss of rest, and he becomes anæmic and thin.

The course of chorea is chronic. It rarely terminates before the end of six or eight weeks, and it often continues for three or four months. In rare instances it becomes habitual, and lasts throughout life. Its course either is marked by remissions and exacerbations, or else the malady increases constantly to its acme; then remains stationary for a while, after which it gradually abates. Recovery is the most usual termination of the complaint. The cases are exceptional in which chorea becomes habitual; but it often happens that, in certain of the patient's motions, a trace of the former disease remains for years, as well as a tendency to relapse. Permanent mental derangement is also rare. Death scarcely ever occurs, unless through complications. There are, however, a few instances on record where it has been the result of the disease itself. In these cases the muscular contractions rapidly become extremely violent and general. This was followed by collapse and coma, in which the patient perished.

Treatment.—Our scant knowledge as to the causes of chorea puts a fulfilment of the causal indication almost out of the question. When the disease has been preceded by symptoms of anæmia and hydræmia, the ferruginous preparations are to be selected from the long list of reputed remedies against chorea. We do not attempt to say whether carbonates of iron (gr. v—gr. x pro dosi), or ferr. hydrocyanic (gr. ij—gr. iij pro dosi), which are the favorites, possess any real advantage over the other preparations. When the patient has suffered from rheumatism, the sulphur-baths (as recommended by *Baudeloque*), an ounce of sulphuret of potassium to twelve gallons of water, in which the patient is to spend an hour at a time, are as much to be recommended as are the chalybeates, when there is anæmia. Where worms are known to exist in the intestine, the treatment may be commenced by a dose of santonin or other anthelmintic.

The number of remedies proposed for the cure of the disease itself is very large. As, however, the disease usually subsides spontaneously, in the course of six or eight weeks, and as it very rarely is possible to cut the malady short in a less period of time, we are very apt, if quite candid and circumspect in measuring our success in special instances, to be in doubt as to whether the chorea abated in consequence of a six weeks' administration of the medicine prescribed, or whether it "got well of itself." Fortunately, most of these remedies, if cau-

tiously given, can do no harm. This is especially true of the preparations of zinc, particularly of the oxide. The sulphate, the valerianate, and the hydrocyanate of zinc, are less innocent, but not more to be relied upon, nor are the ammonio-sulphate of copper and nitrate of silver. *Romberg's* valuable opinion is in favor of arsenic; and, indeed, where we have determined to employ the metallic nervines, *Fowler's* solution (gtt. iij—gtt. v t. d.) is to be preferred to all others. The narcotics may be dispensed with, as a rule, in treatment of chorea, and, moreover, are apt to be so ill-borne that we often have regretted the administration of an evening dose of Dover's powder or morphine to insure the rest of the patient. My experience as to the action of opiates is, however, in opposition to that of one of our great authorities. *Trousseau* urgently recommends the administration of large doses of morphine, and claims, too, that they are remarkably well borne. Strychnine, another medicine for chorea, strongly recommended by *Trousseau*, and which has been given, as directed by him, at first in very small doses, afterward in larger ones, until slight signs of poisoning set in, has met with little approval in Germany. Their introducer's own experience, which by no means resulted in sudden or even in rapid cures, offers no encouragement to make use of these noxious remedies. This is also true of the proposed hypodermic injection of curare. With this article the certainty of benefit is by no means proportionate to the dangers incidental to an overdose. Whenever the vertebræ are sensitive to pressure, a few leeches or cups may be applied by the side of the spine, to be followed by counter-irritants to the skin. Care should be taken, however, not unnecessarily to disfigure the necks of young girls by scars; hence we should avoid the use of tartar-emetic ointment. Cold affusion upon the back, which is probably the best method of producing determination to the skin, seems to be of decided benefit in some cases, especially in cases of long standing, although it sometimes aggravates the disease. The inhalation of chloroform is an excellent palliative for the severer forms of the chorea; but further experience only can determine whether the repeated and long-continued exhibition of this article, pushed to the point of complete narcotism, has the effect of abbreviating it. A more careful trial will be requisite ere the general adoption can be sanctioned of the practice of forcibly holding or tying patients, a practice which has been proposed by several authorities. *Benedikt* declares that, "out of more than twenty cases of chorea, treated by him by the constant galvanic current, not one has failed to recover." The current which he employs is just strong enough for the patient to feel it distinctly, and he applies it along the spine, the patient standing erect. Painful currents aggravate the symptoms. During convalescence we

should endeavor to teach the patient, by a systematic and humane system of discipline, to resist the involuntary motions by the force of his will.

CHAPTER II.

LOCKJAW—TRISMUS—TETANUS.

Etiology.—Like chorea, lockjaw is a derangement of the motor function. The symptoms of this disease are ascribable to a morbid excitement of the motor nerves, the participation of the sensory nerves in the disease being but slight, and, for the most part, of a secondary character. In this malady, however, we know, with much greater certainty than we do in chorea, that the morbid irritation of the motor nerves proceeds from the spinal marrow. This hypothesis is supported rather than contradicted by the fact that the results of *post-mortem* examination of the spinal marrow usually are negative in cases of tetanus, and that real tetanic spasms are seldom observed in cases of grave organic disease of the spinal cord. It would be quite impossible for motor impulses to originate from a spinal marrow reduced to a mass of *débris*, or whose elements were otherwise degenerate or destroyed, while experience teaches that the lesions from which abnormally-active impulses proceed are insusceptible of anatomical demonstration. At the outset of the disease, tetanic spasms are generally produced by the action of trifling but still appreciable irritants, which, acting upon the extremities of the peripheral nerves, throw the spinal marrow into a state of excitement, so that, at this period, the spasms, although distinguishable from other reflex symptoms by their greater violence and longer duration, may be called reflex spasms. As the malady advances, however, such causes are not required in order to give rise to the cramps. The spinal marrow then remains permanently in the condition of intense excitement into which it is thrown by the motor nerves.

With regard to the etiology of the disease, a number of noxious agents may be enumerated, which can be proved capable of inducing the morbid state of the spinal marrow to which tetanus is due. First among these are wounds, especially lacerated, punctured, and gunshot wounds, and wounds in which foreign bodies remain lodged. Injuries of this kind are more dangerous upon the extremities than upon other parts of the body; but they never give rise to tetanus except under certain conditions, of which, sudden change of temperature (such as hot days followed by cold nights) is known to be one, while others are unknown. *Bardeleben* aptly sums up this condition as follows: that the wound is the predisposing agent, and the chilling the exciting cause. We do not know, however, what the changes in the nerve-

tissues are which induce tetanus, nor how they affect the spinal marrow. The injection and swelling in the course of the nerve, between the wound and the spinal marrow, which have sometimes been observed, are by no means constant. In other cases, lockjaw occurs from the effect of cold, without any previous wound, as when a man has slept upon the moist earth, or been wet while his body is heated. This form of rheumatic tetanus is much more rare than the traumatic form. Here, too, we are completely in the dark as to the ways and means by which the lesion, suffered by the cutaneous nerves in rheumatic tetanus, is transmitted to the spinal marrow. The tetanus observed sometimes in newly-born infants (tetanus neonatorum) is usually regarded as a third form of the disease, although it should properly be reckoned as belonging to the traumatic variety. Tetanus neonatorum never occurs excepting between the first and the fifth day after the fall of the navel-string. Hence it has always been immediately preceded by a wound (ligation and binding of the cord). The objection that, in some cases, there is inflammation of the umbilicus, while in others there is none, is not valid; for, even in the wounds of adults which have caused lock-jaw, although, in most cases, they are violently inflamed, and otherwise affected, yet there have been instances in which the wound was doing perfectly well, and was healing, or even was completely cicatrized. Besides, in infantile tetanus, the ligation and division of the umbilical cord can only be considered as the remote cause, while the chilling of the skin, or other unknown agent, which sometimes seems to be of an epidemic nature, must be regarded as the immediate cause. Finally, a morbid condition of the spinal cord is induced by poisoning with strychnine, which exhibits precisely the same symptoms as those of lockjaw, so that the toxic signs induced by strychnine and brucine have been called tetanus toxicus.

Traumatic and rheumatic tetanus is of far more frequent occurrence in men than in women. Vigorous constitutions are more liable to it than feeble ones. In the tropics the disease is more common than with us; and certain races, especially negroes, seem to be more prone to the disease than Europeans.

Symptoms and Course.—Tetanus is characterized by continuous tonic spasms, which principally involve the muscles of the trunk and those of mastication, and which are marked by paroxysmal periods of aggravation of extreme intensity.

The malady is usually preceded by signs consisting of a febrile condition of no great severity, and pain, and stiffness in the back of the neck, which may readily be mistaken for a trifling fit of rheumatism. If such symptoms make their appearance after receipt of a wound of the character above given, and if at the same time the

aspect of the wound change, and should it become dry and painful, we may already fear great danger to the patient. When the disease has fairly set in, the head at first is almost always fixed and drawn backward, by rigid contractions of the muscles of the neck. Tonic spasms of the muscles of mastication press the jaws firmly together (lockjaw, trismus), and at the same time deglutition is impeded or prevented by spasm of the pharynx. From the nape of the neck the disease extends over the muscles of the back. Thus the entire body is bent backward in the shape of a bow. The abdominal and thoracic muscles, however, are also involved in the spasm. Hence the belly is tense, contracted, and hard as a board, and there is a sense of constriction at the pit of the stomach, which is very painful to the patient. More rarely the muscles of the extremities, particularly those of the forearms, legs, hands, and feet, are attacked by cramps. The terms *opisthotonos*, *emprosthotonos*, and *pleurosthotonos*, are severally applied to the disease, according as the contractions preponderate in the posterior muscles of the neck and back, in the anterior muscles of the neck and trunk, or in those of the side, and according as the body is drawn forward, backward, or sidewise. When there is no such preponderance, so that the body lies rigid as a statue, the condition is called *orthotonos*. Opisthotonos is by far the most common form of the malady; the other varieties are rare. The contracted muscles remain upon the stretch throughout the whole disease. From time to time, however, paroxysms occur, in which the cramps are so severe that the muscles sometimes are torn asunder. At such times the middle of the body is suddenly jerked into the air, so that no part of it touches the bed except the head and heels. The muscles are hard as stone, and are the seat of frightful pain, which can generally be compared to that of a severe cramp of the leg. The contours of the temporal and masseter muscles stand out in bold relief, imparting a peculiar expression of countenance. This is still further increased by contractions of the mimic muscles of the face. The forehead is wrinkled, the brows knit, the eyes rigidly fixed, and sunken deep into their sockets. The angles of the mouth are drawn outward, and the lips are drawn apart, exposing the clinched teeth. The muscles of the extremities are affected last; this is an important point in diagnosis. At the outset of the disease the paroxysms do not occur spontaneously, but are provoked by the most trifling causes. Just as in a frog poisoned by strychnine, in which tetanic spasms may be induced by merely tapping upon the table on which he lies, so the slightest touch upon the skin or breath of air upon it, a faint jolt of the bed, the sound of shutting a door, every movement which a patient desires to make, the acts of chewing or swallowing, or even the suggestion of the idea, suf-

fice to bring on a new attack. The inability to swallow, and the spasmodic seizures induced by every effort to drink, create a certain resemblance between tetanus and hydrophobia. The paroxysms are of variable duration. At first they are brief, but at the height of the disease they may continue for a quarter of an hour, or even an hour, ere the remission set it. Consciousness and the intellect generally remain unimpaired until the last in this frightful malady, nor are many of the other functions materially deranged. The unhappy patient suffers from hunger and thirst, which he is unable to relieve. As in all other violent muscular exertion, the skin is bedewed with sweat, and the pulse is frequent and small. The enormous elevation of the temperature, first pointed out by *Wunderlich*, is a matter of great interest. Now and then it has been observed as high as 110° F., and immediately after death a further rise has been observed almost up to 112° F. [This may be due to the muscular activity. *Leyden* and others have shown that the same elevation of temperature occurs in tetanus artificially induced in dogs. But similar rises are said to occur after injury of the cervical medulla and other cerebral and spinal diseases of the warmth-producing centres; as in *J. W. Teale's* case, where the temperature was 108°–122° for some weeks, as witnessed by several physicians.] The bowels are usually somewhat constipated, and sleep, though ardently longed for, is impossible. Another source of anguish, as well as of the greatest danger, consists in the derangement of the respiration. There is no obstacle to the entrance of the air, but, as *Watson* aptly remarks, the chest is compressed as in a vice, and respiration is made extremely difficult by reason of the rigidity of the muscles. There are instances where the victim perishes only a few hours after the outbreak of the disease. In such cases, the spasmodic contraction of the diaphragm and other respiratory muscles so increases as to completely arrest respiration and cause suffocation. The majority of patients are not relieved from their sufferings so soon. For three or four days, the cramps, and the frightful pain and dread of suffocation which accompany them, continue to increase in duration and frequence, the remissions becoming more and more imperfect; until the sufferer expires, either poisoned by carbonic acid resulting from the imperfect manner in which respiration is carried on, and from the augmentation in the consumption of the oxygen, or else dying during some very severe paroxysm, through sudden and absolute interruption to the respiration. Sometimes the breathing is not so much interfered with as to cause death from lack of oxygen. The disease may then go on for weeks ere the patient, in a state of extreme emaciation, and exhausted by privation of nourishment, succumbs to starvation. Recovery is extremely rare. Transitory remissions, in which the

patient may even enjoy a brief refreshing slumber, should not be allowed to deceive us. After such pauses the malady generally breaks out with all its former severity, or even with increased violence. We must not indulge in the hope of recovery unless the seizures continue to diminish in length and frequence, distinct relaxation of the contracted muscles occurring during the intervals, and unless the patient become able to take food and nourishment. Even in the most fortunate cases this takes place with extreme slowness; and it is not until after the lapse of weeks that the muscles entirely lose their tension, and the patient is completely well.

The symptoms of tetanus neonatorum are but little modified by the peculiarities of the infantile organism. Here, too, the outbreak of the disease usually is heralded by indistinct prodromata. The child cries out frequently during sleep; has blue rings around the eyes and lips; and lets go the breast which it has just seized with avidity. The mother first becomes aware of the onset of the disease itself when she finds that neither the nipple nor the finger can be inserted into the mouth. The jaws stand several lines apart, but it is impossible to separate them further. The malady increases rapidly. The muscles of the face are also contracted spasmodically. The forehead is wrinkled, the eyelids firmly shut, and surrounded by converging wrinkles. The alæ nasi are dilated, the lips compressed and puckered, while the tongue usually is fixed between the jaws. Besides this, there is opisthotonos, the head is drawn backward, and the spine arched like a bow. A touch, or an attempt to move or swallow, provokes a violent spasm; but the remissions usually are more complete than they are in the tetanus of adults. Respiration is impeded, during the seizures, by the rigidity of the thorax and the tension of the abdominal muscles. Attacks of suffocation arise, of which the child often dies in from twelve to twenty-four hours. In other cases the breathing gradually becomes insufficient, and the child succumbs to overcharge of the blood with carbonic acid. Death then takes place more slowly, but still within a few days, during which the child loses flesh rapidly. Recovery from tetanus neonatorum is likewise extremely rare.

Treatment.—For the fulfilment of the causal indication, a series of surgical operations—even amputation of the wounded member—have been proposed and practised. The success of these operations, however, was by no means what had been anticipated of them, and more recently they have been abandoned. From the prominent part which is unmistakably played by exposure to cold, both in rheumatic and in traumatic tetanus, warm and stimulating baths, as well as Russian vapor-baths, would seem to be indicated. The objection, that the manipulation necessary in giving such baths tends to aggravate the

symptoms, only held good in two of *Hasse's* cases, and that only at the very commencement, and not after the manipulation had been continued. This circumstance and the relief which the baths afford to the patients imperatively demand their employment.

Upon the supposition that tetanus was an inflammatory affection of the spinal marrow, the attempt was formerly made to fulfil the indication of the disease itself by blood-letting, local and general, and by the exhibition of calomel to the point of salivation. Such procedures, however, have been abandoned more and more in later times. Unfortunately we do not possess any remedy capable of bringing back the irritable state of the spinal cord to its normal condition. Even the narcotics do not have this effect, although they are indispensable for the purpose of alleviating the sufferings of the patient. To accomplish this, large doses must be given; so that, unless the approach of narcotism be vigilantly watched, there is danger of hastening the fatal termination. If the patient cannot swallow, morphia should be given hypodermically, or else clysters, containing twenty or thirty drops of laudanum. Injections of tobacco are not of much greater service than opium-clysters, and, unless the dose be regulated with extreme caution, they are much more liable to give rise to a fatal collapse than the latter. The anæsthetics, if possible, are of still greater importance than the narcotics; but, unfortunately, their effects likewise are merely palliative. Too free use of them must be avoided, and the patient must not be kept in a state of permanent insensibility from chloroform. The English recommend the stimulants, especially carbonate of ammonia, brandy, and wine, to which they give much greater credit than to blood-letting and the narcotics. Trustworthy observers have obtained very great benefit from the hypodermic injection of a solution of curare in tetanus. This treatment certainly deserves further trial. However, owing to the very variable character of the curare, it will be necessary, prior to the exhibition of the preparation to be used, to ascertain, by experiment upon animals or upon the healthy human being, how large a dose may safely be administered. When this precaution has not been taken, we must commence with very small doses (gr. $\frac{1}{8}$ to gr. $\frac{1}{4}$), and gradually increase them to gr. $\frac{1}{2}$ to gr. $1\frac{1}{2}$. *Demme* advises that a solution of one or two grains in one hundred drops of water be employed, ten drops of this to be injected as a dose. According to *Demme*, the action of curare lasts four or five hours, and then begins to abate; and upon this fact the repetition of the dose may be regulated. It is most important that the patient should be kept in a quiet chamber, with a uniform temperature, and that his eyes should be screened from too bright a light.

For the tetanus neonatorum we may prescribe camomile-baths,

clysters containing a drop of laudanum, and, if the spasms be very severe, chloroform may be administered, but with great caution.

CHAPTER III.

EPILEPSY—FALLING SICKNESS—MORBUS SACER—HAUT-MAL.

Etiology.—Unlike chorea and tetanus, epilepsy cannot be called a purely motor neurosis; since the interruption which takes place, both in consciousness and insensibility, is quite as essential an element of an epileptic fit as the convulsions. The absence of one or other of these symptoms renders the seizure imperfect.

We may assume that the excitement of the motor nerves, of which the convulsions are the exponent, proceeds from the medulla oblongata and the portion of the brain lying upon the base of the skull. This is shown.

1. By the interruption of the functions of the hemispheres, which accompanies the convulsions. It is not probable that motor impulses proceed from the hemispheres at a time when the irritability of the other ganglion-cells and nerve-fibres is extinguished.

2. Because convulsions, similar to epileptic convulsions, can be excited by continuous excitement of the basilar portion of the brain by means of the induction apparatus, while no such result is obtained by a like irritation of the various parts of the hemispheres.

3. Because *Küssmaul* and *Tenner*, in their experiments upon animals, could still produce convulsions of a decidedly epileptiform character after extirpation of both hemispheres.

4. *Schroeder van der Kolk* has found that, in all bodies of epileptics where the disease had been of long standing, besides numerous inconstant lesions, there was alway a dilatation of the arterioles and capillaries of the medulla, with thickening of their walls. The influences which tend to produce this condition of the medulla oblongata, whereby it throws the nerve-fibres passing through it or originating from it into such intense excitement, and which, for brevity's sake, we call a state of irritation, are probably of a manifold character. It is true that the experiments of *Küssmaul* and *Tenner* have proved that epileptiform convulsions may be induced by cutting off the supply of arterial blood from the brain; but they do not prove that arterial anæmia of the brain is the sole cause of epileptic fits. *Schroeder van der Kolk* believes that epileptic convulsions depend mainly upon an increased afflux of arterial blood to the medulla oblongata. There is no doubt, moreover, that the morbid irritability of the medulla which occasions epilepsy may arise without any increase or diminution of its

supply of blood, merely from the improper character of its nutriment and from the admixture of certain materials in the blood. It must also be admitted that the medulla may be thrown into an irritated condition by the transmission of a morbid impression from remote regions of the nervous system, whether central or peripheral. It is well known that neuromata and cicatrices, or tumors pressing upon peripheral nerves, have sometimes occasioned epilepsy (although, indeed, such instances are not common), and that the epilepsy has ceased after section of the affected nerve or after removal of the cause. Perhaps, also, cerebral tumors and other diseases of the brain and spinal marrow may induce epilepsy in a similar manner by gradual transmission of a morbid irritability to the medulla oblongata. This supposition has received strong support from the result of recent experiments by *Brown-Séquard*, in which dogs, whose spinal marrows had been injured, suffered from convulsions, although not immediately, but some time after the receipt of the injury. It is difficult to say why the morbid irritability of the motor nerves is not continuous, but merely occurs in paroxysms, with intervals which frequently are of very long duration. Are we at liberty to suppose that it is only now and then that the medulla oblongata falls into this morbid state? Is there really ground for the hypothesis that the cause of epilepsy is a transitory spasm of the muscular fibres of the arteries, with consequent arterial anæmia? Is it true that poisons, or the irritation of remote tumors, or other agents which give rise to epilepsy, act by the occasional provocation of the spasm of the muscles of the blood-vessels? May we, with *Schroeder van der Kolk*, compare the ganglia of the medulla oblongata to a Leyden jar, or to the electric organ of certain fishes? May an epileptic fit be likened to the spark from the Leyden jar, or to the discharge of the electric organ of the electric fish? and do the ganglia reload themselves, as it were, for a fresh explosion during the intervals? Or, finally, is the morbid state of the medulla constant, but requiring the additional stimulus of some new transitory irritant transmitted to it from some remote point in the brain, the spinal cord, the peripheral nerves, or the intestines, in order that the fit may occur? Of all these conditions we as yet have no accurate knowledge, and it were idle to advance other hypotheses in explanation of the foregoing ones.

Equally inexplicable is the constant coexistence of irritability of the spinal marrow with palsy of the cerebral hemispheres. The arrest of sensation and consciousness has sometimes been regarded as a secondary condition, resulting from the convulsions. It has been attributed in part to venous engorgement of the brain, arising from compression of the cervical veins by the contraction of the cervical mus

cles, and in part to surcharge of the blood with carbonic acid, in consequence of spasmodic closure of the glottis. The fact that the loss of consciousness and sensation and the occurrence of the convulsions are almost always simultaneous; and that the former sometimes precedes the latter; and that in many cases of incomplete epilepsy it is the sole symptom of the seizure, is a sufficient refutation of both hypotheses. In like manner it must be declared that there is no proof as to the truth of the theory of *Schroeder van der Kolk*, that, in an epileptic fit, the ganglion-cells of the medulla excite a spasm of the vasomotor nerves of the brain, simultaneously with that which they induce in the cerebro-spinal nerves, and thus cause cerebral anæmia and palsy. So, too, with *Henle's* theory, which supposes the existence of a plethoric and an anæmic form of epilepsy. He suggests that, in the former, besides the more intense hyperæmia of the hemispheres which induces the palsy, a lesser degree of hyperæmia, capable of inducing symptoms of mere irritation, arises in the medulla oblongata; while, in the anæmic form, he imagines that the lack of blood in the cerebral vessels occasions an increased blood-pressure upon the medulla oblongata, inducing in it a degree of engorgement sufficient to cause signs of irritation. Here, too, we shall refrain from further theorizing, preferring to admit that to us the antagonism existing in epilepsy, between the condition of the hemispheres and that of the basilar portion of the brain, is entirely inexplicable.

Owing to the scantiness of our knowledge as to the pathogeny of epilepsy, the statistics regarding its predisposing and exciting causes are of but secondary importance. We do not know of one single agent of which we can certainly predict that it will produce epilepsy; yet, besides this, we must admit that all the assigned causes of this disease by themselves are incapable of inducing it, and that the cooperation of a second and unknown factor is always requisite. Statistics show epilepsy to be a very common disease, about six epileptics being found in every thousand individuals. Females suffer from it somewhat more frequently than males. There is no age which is completely exempt from it; but the majority of cases occur between the tenth and twentieth years of life; next to this, between the second and the tenth year, and between the twentieth and thirtieth year. It seldom commences during old age, and its congenital occurrence, as well as its appearance in the first months of life, is equally rare. Hereditary predisposition plays an unmistakable part in its production; its existence is demonstrable in nearly a third of all cases. It is especially apparent in persons descended from epileptic parents, particularly from epileptic mothers, as well as in individuals whose parents or ancestors have been insane or intemperate. In some families

epilepsy afflicts many of its members throughout several generations. Sometimes the disease skips a generation, and the grandchildren are attacked, but not the children. Cachectic subjects, drunkards, and onanists, are more liable to it than healthy, vigorous persons, although the latter are by no means exempt. The chief of its exciting causes are violent mental emotions, sudden fear, terror, and the sight of an epileptic fit. In more than a third of all cases the first attack has followed upon some violent fright. The most common structural alterations found in the skull and brain, which, however, like the mental emotions, are not constant, and only give rise to epilepsy under certain unknown conditions, are asymetry, imperfect development of the skull, diffuse thickening or exostosis of the skull, thickening, adhesion and ossification of the dura mater, tumors and deposits in the brain, chronic hydrocephalus, and cerebral hypertrophy. Alteration in the appendages of the brain, which *Wenzel* mentions as a constant lesion in epilepsy, is absent in the majority of cases. Changes of structure in the spinal marrow are not so frequently found in epilepsy as similar changes in the brain; but perhaps this is because the former have been less diligently examined than the latter. We have already alluded to the neuromata, tumors, and scars, which sometimes give rise to the disease by the pressure which they exert upon peripheral nerves. In a similar manner, epilepsy may arise from an abnormal irritability of the sensory nerves, induced by some severe irritation at their peripheral extremities. Thence, according as the irritation involves the nerve-tips of the thoracic organs, those of the organs of digestion, or those of the urinary or sexual apparatus, the epilepsy is classified into epilepsia cardiaca, pulmonalis, abdominalis, nephritica, uterina, etc. It is manifest that we may easily err in attributing the disease to irritability of one or other of these organs. Uterine epilepsy is perhaps the least ambiguous of all, as a gradual transition from hysteria to epilepsy may sometimes be observed, and as some women become epileptic upon their first coitus. The presence of worms in the intestines also is sometimes an unmistakable cause of the disease.

Symptoms and Course.—Epilepsy is a chronic disease, characterized by convulsive attacks, accompanied by loss of consciousness, with intervening periods of exemption of variable and sometimes of very long duration. Loss of consciousness during an epileptic fit necessarily involves loss of sensation and incapacity for voluntary motion. In incomplete epilepsy, the "petit mal" of the French, there usually are no convulsions during the seizure, or else merely a few twitchings. Such rudimentary attacks, of course, are not to be called incomplete epilepsy unless they alternate with well-pronounced seizures, or arise

from them, or unless, in the further course of the case, the rudimentary fits gradually developed into perfect ones.

In some patients an epileptic fit is regularly, or at all events generally, ushered in by an *aura*. This *aura* receives its name from a sensation as of a vapor which rises from the extremities toward the head, and terminates in the fit. This prodromic feeling, however, is only described by a few patients. Far more frequently there are other sensations, such as a sense of creeping, of warmth, or numbness, or a peculiar pain in some part of the body darting thence to the brain, which herald the attack, and which are called the "aura epileptica." Instead of this sensible signal, the fit is preceded in other instances by twitching or palsy of some part of the body. This is called the motor aura, in contradistinction to the sensory aura above described. In other cases, again, the seizure is ushered in by abnormal phenomena in the organs of sense, hallucinations, visions of sparks or of colors, buzzing in the ears, the report of a loud crack or other sound, dizziness, and sometimes by the regular recurrence of phantasmagoria of more or less grotesque character. This latter form of sensorial or mental aura by no means proves that the epilepsy is of central origin, in the sense, at least, that the malady is the result of appreciable lesion of the brain; nor is the occurrence of an aura arising from the extremities to be regarded as a proof of the peripheral origin of a case of epilepsy. It is an extraordinary fact that an epileptic fit may sometimes be averted by binding a ligature firmly above the starting-point of the aura, and thus, as it were, isolating it. That even such a phenomena as this is no proof of the peripheral origin of an epilepsy is in some degree manifested by the experiments above mentioned of *Brown-Séquard*. Here it was demonstrated that, in the dogs made epileptic artificially by wounding of the spinal marrow, a fit occurred wherever the skin was irritated within the province of a particular branch of the trifacial nerve. We cannot tell whether the fit was preceded by an aura in these dogs, but we may positively conclude from these experiments that, even when the individual epileptic attacks are induced by peripheral irritation, the real cause of the disease may consist in palpable structural disease of central organs.

Whether preceded by an aura or not, the outbreak of the paroxysm is usually announced by a shrill cry, whereupon the patient loses all sense and falls to the ground, usually backward or sideways. He scarcely ever has time to seek a convenient place, but falls, regardless of place, often in the most perilous situations, striking, perhaps, against a hot stove, a sharp corner, or down the stairs. There are but few epileptics, whose disease is of long standing, who do not carry with them the marks of more or less severe injury. The fall is usually fol-

lowed at first by tonic contractions, in which the body and extremities are extended, the head drawn backward or to one side, the mouth firmly closed, the eyes wide open, and rolled upward or inward, the thorax fixed, and the respiratory movements arrested. After a few moments, during which the jugular veins become distended and the face purple, the tonic spasms are converted into clonic ones, which soon convulse the whole body. The countenance, hitherto immovable, is now thrown into active agitation; the angles of the mouth are drawn hither and thither, the forehead and eyebrows twitch, the eyes open and shut, the jaws are forcibly pressed together, and are worked backward and forward, so that the teeth grate audibly. The teeth are not unfrequently broken off, the tongue bitten through, and even the lower jaw may be luxated. Upon the lips there appears a saliva, rendered frothy by the constant movements of the mouth, and which, too, is often bloody from wounds of the tongue or cheeks. The head is jerked forward and backward, and from side to side; while the convulsive twitchings of the muscle of the trunk pitch the body hither and thither. In the extremities, and especially in the upper extremities, quick kicking, striking, twisting, and twitching motions occur in turn, and with such violence as sometimes to result in luxation or fracture. The fingers usually are flexed, the thumb being pressed into the palm of the hand, a sign which the laity erroneously believe to be pathognomonic. Sometimes it seems as if the violence of the convulsions were remitting, and as though the fit were about to abate; but the lull is soon followed by a fresh outbreak, and the convulsions become more violent than ever. Sometimes the twitching is superseded, for a few moments, by a tetanic condition like that by which the paroxysm commenced. Throughout the whole fit the respiration is much embarrassed, owing to the impediment offered by the tonic or clonic spasms of the respiratory muscles to the regular heave and fall of the chest. Still more is this the case when the glottis is closed by spasm of the laryngeal muscles. As on all other occasions of unusual muscular exertion, the beat of the heart is accelerated, the pulse is small and sometimes irregular, while the skin is bathed in sweat. The bowels and bladder are often evacuated unconsciously; more rarely erections and seminal emissions occur. Throughout the entire seizure, consciousness is so completely extinguished that the patient is not aroused, nor does he betray any sign of pain, even though he may strike against a red-hot stove, or fall into the fire so as to completely char a limb. Opinions are divided as to the state of the reflex action during the attack. I admit that I find it very difficult to take note of it in the more violent class of fits. During seizures which are mild from the beginning, or during the period of subsidence of more

severe attacks, I have been able fully to confirm *Romberg's* observations, that the reflex action is not arrested. The patient closes his eye if the conjunctiva be touched, and winces if his face be sprinkled with cold water. One observation of *Hasse's* is exceedingly strange and difficult of explanation. In a patient of his he found the electric contractility was extinguished in the muscles of the thorax and extremities. After the fit has lasted for ten minutes, or, at most, for a quarter of an hour (although it may seem much longer to the by standers), either it subsides, the twitching gradually growing weaker and weaker, and finally ceasing, or else it may stop suddenly, the convulsions being all at once followed by complete muscular relaxation. The fit often terminates by a long, sighing expiration; more rarely by vomiting, by passage of gas upward or downward, or by a profuse evacuation of the bowels.

It rarely happens that a severe epileptic seizure is immediately followed by complete recovery; after the attack is over, generally, unless violently roused, the patient falls into a deep sleep, with prolonged stertorous respiration. If awakened from this, he usually looks blankly or anxiously around him. He does not know what has happened, and can scarcely account for his being in a strange place, or in bed, or wounded. His sole desire is to be allowed to sleep. Next morning, though still somewhat unwell and low-spirited, and complaining of confusion about the head, he has recovered his faculties, and all traces of the attack vanish in course of the day.

Many epileptic fits differ considerably from the typical seizure above described, varying in duration, violence, and in the extent of the convulsions. The variation of the condition which immediately succeeds the paroxysm is of more importance. While, as a rule, the patient is able to attend to his business the day after his fit, there are instances in which every attack, or else each severe one or rapidly recurrent succession of them, is followed by mental derangement, and other disorders of innervation. The former may consist of well-marked fits of mania. It is often necessary to protect both patient and bystanders from violence, to put on a strait-jacket, or even—horrible to relate—there is no other resource but to confine the patient in the mad-house, even during his lucid period, because it is known that his madness will recur with his next paroxysm. In other instances the fit is succeeded by a condition usually called partial insanity: for example, the patient may have an irresistible inclination to run. In other cases, the patient may be in an irritable mood, which is quite foreign to him on ordinary occasions, breaking out into violent rage upon the slightest provocation. Sometimes the memory and mental acuteness are impaired for some days. We may also mention that, in

stead of the latter condition, an opposite one has been reported by certain observers, the intelligence of the patient becoming remarkably acute. With regard to the other disturbances of innervation, we have already alluded to the transient and permanent paralysis of the extremities, which perhaps is due to exhaustion of the irritability consequent upon excessive nervous excitement. The fit may also be followed by aphonia, by dysphagia, or by asthmatic attacks, etc.

Two forms of the incomplete epilepsy are recognized (especially in France), slight convulsive movements accompanying the lapses of consciousness in one, and being absent in the other. In the former, the genuine "petit mal," the patient is seized with giddiness, often amid business or conversation. He has time, however, to seat himself, or staggers, and sinks slowly to the ground, without outcry. His face is pale, his eyes fixed. A few convulsive twitches play over the face, and the extremities, especially the upper ones, are slightly tremulous. In a few minutes, however, the patient recovers; looks wildly around him, not knowing what has happened, sometimes making confused utterances; then, after the lapse of a few minutes, all perturbation subsides; there is no somnolent stage, and the patient is once more in a condition to resume his vocation.

In the still milder and more rudimentary form, the *vertige épileptique*, the patient does not fall. His consciousness alone is clouded; his eyes stare, and his countenance is pale. He does not twitch, but lets fall whatever he may have in his hand, and halts in his speech. The attack passes off in a few seconds, and the patient proceeds with his conversation or business, as if nothing had happened. There are many intermediate stages between epileptic vertigo, the *petit mal*, and complete epilepsy, a description of which we forego.

It would be quite difficult to furnish a concise account of the general course of the disease, and of the condition of the patient between the paroxysms. We have stated already that epilepsy is a chronic affection. Hence cases in which one epileptic fit is not followed by a second one ought not to be counted as epilepsy, but rather should be regarded as belonging to the eclampsia, a form of disease only distinguishable from epilepsy by the acuteness of its course. The pauses which intervene between the fits differ greatly in different individuals. In some patients, a year or even several years elapse, and, in many, months and weeks pass away, ere a new attack occurs, while in other patients one or more fits occur daily. Very often, during a period of four or six weeks, there is not a single attack; this is followed by a series of fits, recurring at short intervals. As a general rule, however the pauses between the paroxysms in the same individual remain tolerably uniform; although, as the malady progresses, especially in young

persons, the fits gradually come closer together. Perfect regularity in their sequence is never seen, or but for a short time; although in women a periodical type is sometimes approximated to when the seizures only occur at the periods of menstruation. In some persons the attacks are more frequent during the day; in others they occur chiefly at night. Nocturnal epilepsy is considered to be of an especially malignant and obstinate character. Generally speaking, it is impossible to detect the exciting cause of the fit; but, besides the fits which occur spontaneously, there are always some which are unmistakably ascribable to psychical emotion, especially to fear; others which may be traced to onanism, to coitus, and, as we have already stated, to menstruation. Sometimes we can tell, from the altered mood of the patient, and from his complaint of a sense of weight in the limbs, headache, or dizziness, that an attack is coming on. In the outset of the disorder, the seizures are usually of the complete form; as it advances, and especially in inveterate cases, both complete and incomplete paroxysms occur. It more rarely happens, instead of this, that the attacks are incomplete in the beginning of the disease, and afterward develop into complete ones as it advances. As, in some cases, each fit is followed by temporary mental aberration, so, too, inveterate epilepsy often results in permanent and incurable insanity—either mania, confusion, or idiocy. But, besides the large number of epileptics who close their lives in the insane asylum, the "morbus sacer" (which the ancients ascribed to the wrath of the gods), in course of time, nearly always completely changes the mental and physical habit of the patient. Acuteness of judgment is lost; memory and power of imagination diminish; the gentler and nobler impulses recede more and more; while the excited and unbridled propensities, lasciviousness and gluttony, often impel the patient to commit violent or criminal actions. They often avoid society, are odd and capricious; are very troublesome to those around them, and are apt to burst into violent fits of anger. The personal appearance of the patient also undergoes a change in cases of long-standing epilepsy. *Esquirol* calls attention to the coarseness of the features of an epileptic, to his swollen eyelids, his thick lips, faltering look, and clumsy body, remarking that this malady deforms the most beautiful countenance.

With regard to the termination of epilepsy, recovery must be regarded as rare, in spite of the opposite opinion of many observers, especially *Herpin*. The more distinctly the malady is traceable to hereditary tendency, and the more plainly it depends upon structural disease of the brain; the longer it has lasted, the more violent the fits, the more frequent their recurrence, and the deeper the impression which they leave behind them, so much the less is the chance of re-

covery. Such a termination appears to occur somewhat more frequently among women than among men; and it is likewise more common among children and old persons than among those in the prime of life. We must beware of building our hopes too sanguinely upon the long-continued intermission of the fits, as this disease very rarely terminates with one violent paroxysm. A better sign is when the fits not only grow rarer, but feebler, and when a distinct change is taking place, both in the physical and mental condition of the patient. In some women, but not in all, there is an intermission of the fits during pregnancy. Such an intermission, however, is nearly always observed during the course of acute febrile disease. Sometimes a permanent cure dates from the period of invasion of some acute intercurrent malady, from the appearance or cessation of the menses, or from the occurrence of some violent mental emotion. It is said that the exanthematous eruptions, and the breaking out of old ulcers, sometimes have a similar effect. But, although epileptics seldom recover fully from their disease, and although they are not often long-lived, yet they rarely die during an epileptic fit, in consequence of suspended respiration, or of extravasation of blood into the brain, or of general paralysis during the comatose state which follows the fit. Their death is much more frequently due to the progress of cerebral disease, which has given rise to the epilepsy, or to the effects of injuries which the patient has received during his paroxysms, and, oftener still, he dies of some acute or chronic affection, which is in nowise connected with the epileptic disease.

We shall have more to say regarding the difference between epileptic and hysterical convulsions when we come to treat of hysteria. In my opinion no sharp distinction can be drawn between epilepsy and eclampsia, although the convulsions occurring in protracted cases of uræmic intoxication are considered to be eclamptic by some authors and epileptic by others. It is tolerably easy to unmask simulated cases of the disease. The anæsthesia is often enough cleverly imitated, and we must not expect that all malingerers will wince when burnt, pricked, or pinched, although such reaction hardly ever fails to occur when a violent stimulus of this kind is applied to them unawares and suddenly. *Watson's* suggestion, to direct the nurse, in the hearing of the patient, to pour hot water upon his feet, the nurse being previously instructed to pour cold water instead, is both practical and original. Nearly all malingerers keep the fit up too long, and devote too much attention to certain symptoms, which they believe to be pathognomonic, such as turning in the thumbs, foaming at the mouth, etc., which an attentive observer cannot fail to perceive. It is a suspicious sign, when a person who claims to have been epileptic for a

long time has no traces of injury upon the tongue or peripheral parts. Very often the simulation is recognizable because the patient makes a bad imitation of the aura. He usually includes this symptom in his comedy, believing it to be an essential one, and in enacting it often makes a most wonderful exhibition. The surest sign that the fit is not feigned, is the dilatation of the pupil, even upon exposure of it to bright light. This is a token which no impostor can imitate.

Treatment.—As a prophylactic means against epilepsy, *Romberg* recommends that the intermarriage of families, in whom the malady is hereditary, should be avoided. An epileptic mother should never suckle her own child, but should intrust it to a vigorous wet-nurse.

In fulfilment of the causal indication, the treatment of epilepsy is extremely difficult. Apart from the cases whose history gives no clew as to the cause of the malady, and even when our information is comparatively of a satisfactory nature, we can only ascertain a few of the secondary causes; and it is not at all common for the disease to subside after they have been allayed. An epilepsy, whose first outbreak was unmistakably provoked by the presence of worms in the intestine, or by a neuroma, usually persists, although the worms have been expelled, and the neuroma extirpated. If the malady be consequent upon fright, the fits nearly always recur, though the patient be screened from further terrors. However, our poor prospect of success should not deter us from taking account of all causes which may have contributed to produce the malady, trifling though their apparent effect may be. Experience teaches that such practice sometimes, although rarely, leads to the happiest results. In the present uncertain state of our means of treatment of this disease, even such exceptional successes are of importance, and may serve as a guide to our proceedings. It is our duty, in assuming the charge of an epileptic, before resorting to the so-called specifics, so to regulate the external relations of the patient, his habits, and his bodily health, that every suspicious condition, to which the origin of the disease can in any way be ascribed, may be corrected. Thus, as feeble, cachectic persons are more liable to the disease than robust and vigorous ones, all excessive or exhausting mental occupation should be forbidden to the patient, and in its stead a moderate exercise both of body and mind should be advised. Epileptic children must not sit six hours a day on the school-benches; but, if possible, should live in the country, should spend most of their time in the open air, and should take cold baths under proper supervision. We should carefully ascertain whether the patient be addicted to great sexual indulgence, to onanism, or to intemperance in drink; and, where such vices are discovered, they must be opposed with inexorable sternness. When the patient shows signs of anæmia and hy-

dræmia, a nourishing diet, an abode in the open air, wine, and iron, are the proper remedies. If the impoverishment of the blood and cachexia depend upon scrofula, rachitis, or tertiary syphilis, appropriate treatment must be employed. Should there be suspicion of plethora, let the diet be reduced, and made to consist more of vegetables, and cause the patient to drink plenty of water and to exercise freely. On the other hand, avoid general blood-letting; for epileptics, though quite tolerant of medicines (particularly the nauseants), do not bear bleeding well. If a scar, foreign body, or tumor be found pressing upon a peripheral nerve, or if a neuroma be discovered upon it, an operation is indicated. This applies especially to cases in which an aura arises from the affected spot. As positive benefit is sometimes obtained by such operations, the knowledge that in many cases neuromata and tumors are extirpated in vain, should not deter us from operating again. The application of setons and moxas, and the use of pustulating ointment to the nape of the neck, are measures which are much in vogue in the form of epilepsy dependent upon structural disease of the brain and skull. Inunction of pustulating salve upon the scalp, and even trepanning, has also been advised in such cases. We refer to the principles laid down in the second part of this section with regard to this subject. Where a tumor, a spicula of bone, an exostosis, or other disease, encroaches upon the cavity of the cranium, and compresses the cerebral vessels, we can comprehend why trepanning should be of benefit, especially to the convulsions, upon the supposition that the operation affords more room for the brain and its vessels. When there is reason to suspect that the epilepsy is induced by the presence of intestinal worms, anthelmintics are indicated. We must be cautious, however, how we encourage the too sanguine hopes of recovery, in which a patient is too apt to indulge who has discovered joints of tape-worm in his stools.

For other forms of abdominal epilepsy a cure may perhaps be found at the baths of Karlsbad, Marienbad, etc. (*Romberg*). In uterine epilepsy, chronic uterine infarction and excoriation of the os must be treated according to rules already laid down. It is in this form of all others that the causal treatment affords the best results.

In order to meet the indications from the disease itself, *Schroeder van der Kolk* urgently recommends repeated application of cups and leeches to the nape of the neck, followed by blisters, issues, and setons. This author regards the above as the only rational treatment, believing that it alone is capable of reducing the morbid irritability of the medulla oblongata, and of relieving its congestion. He considers that all other remedies tend merely to promote the cure by their action upon the remote cause of the disease, by benefiting any morbid condition

which may exist in the brain or intestines, etc. Whether the theory be right or wrong, the success which *Schroeder van der Kolk's* mode of treatment has attained imperatively demands its adoption. In two very severe but still recent cases of epilepsy I have applied leeches to the back of the neck (four at a time), repeating the application at intervals of from a fortnight to four weeks. The after-bleeding was encouraged by means of elastic cups. The influence of this treatment upon the number and violence of the fits was so brilliant that I could not make up my mind to desist from the blood-letting, and to proceed to the derivatives and medication. If the measures advised in discussing the causal indication, as well as the procedure above proposed, remain without effect, nothing is left but to have recourse to the remedies which are reputed to have a specific action against epilepsy. It would savor too much of pessimism to deny that the commendation awarded to these articles is based upon a certain amount of benefit derived from them, but, unfortunately, it must be admitted that we are quite ignorant as to the conditions under which one or other of them is to be preferred. In this respect the most experienced physician stands upon a par with the tyro. He must try one or other of these remedies, and, if it fail, must try another; and, in this frightful disease, he should never remain idle on account of mere theoretical considerations. With respect to the use of the anæsthetics and narcotics, excepting atropine, we agree with *Schroeder van der Kolk* in condemning them, and for similar reasons. In epilepsy our task is not to allay an exalted sensibility or pain, but to soothe the undue reflex irritation which causes the convulsions. Now, narcotics actually tend to increase reflex irritability, insomuch that in large doses they produce convulsions. Chloroform, too, although it interrupts sensation generally, augments the reflex action. A person under its influence is like a decapitated frog; its reflex movements are more active than ever, although it feels no pain. Atropine is an extremely energetic article, but it seems nearly always to act favorably upon both the severity and number of the fits, even in cases of very long standing. True, I have not seen complete recovery of inveterate cases, and I have never used atropine in recent ones. Moreover, even at a dose not exceeding one-fiftieth of a grain, not only did some of my patients complain of a disturbance of vision, so that they could not do the simplest work, but they suffered for hours with a dryness in the throat which actually prevented them from swallowing solid food. *Trousseau*, who believes belladonna to be the most efficient remedy against epilepsy, gives the following directions for its exhibition with his characteristic thoroughness and precision: Extract of belladonna and the pulv. herb. belladonna, āā ℈j, are to be made into one hundred pills. Of these

the patient must take one daily during the first month; with every succeeding month he is to raise the dose one pill up to five, ten, fifteen, and twenty pills or more. The entire dose is always to be taken at once. When any improvement takes place, the last dose is to be continued for a while, and then gradually reduced in quantity. The prime condition of success is patience on the part both of physician and patient. Instead of the pills, we may use a solution of atropine (atropine gr. ij—in spirit vini. rectif. ʒ ijss), a drop of which corresponds to one of the pills. We, therefore, begin with one drop, and gradually increase to twenty. Of the metallic preparations, those of zinc have the greatest reputation. Latterly, instead of white oxide of zinc, formerly given to the extent of two drachms daily, the salts of zinc have come into use, especially the valerianate and hydrocyanate, and, above all, the acetate. The dose of the latter is twelve grains a day at first; subsequently more. This remedy is said to be most efficacious in young subjects, and in so-called abdominal and uterine epilepsies. *Heim* and *Romberg* recommend nitrate of silver in doses of two to four grains, but it must not be continued long enough to produce *argyria;* ammoniated sulphate of copper and arsenic are now little employed. Of the vegetable nervines, artemisia vulgaris and valerian have the best reputation. Five or ten grains of the former are to be given in powder, or else an infusion of one or two drachms in beer. Of the valerian we at first may give a drachm daily, in powder or electuary, gradually increasing the dose to half an ounce. Quite recently, bromide of potassium has been one of the most generally employed articles in the treatment of epilepsy; many say they have used it with remarkable success, while others report its absolute failure.

My experience of the efficacy of bromide of potash in epilepsy has been greatly increased of late, and I can now speak much more decidedly on the subject than two years ago, when preparing the seventh edition of this work. The following circumstance induced me to use the remedy in as many cases as possible, and to watch the results: I heard that two cases of inveterate epilepsy, that I had for years treated without any benefit, had been completely cured by a so-called specialist, whose advertisements were to be found in the columns of almost every newspaper. I investigated the subject more closely, and found that in one patient, whose governess had for years kept an accurate journal, and whom I had not lost sight of, the attacks had been absent for several months, and that the general health, which had been much impaired, was decidedly better. This "specialist" refused the petitions of numerous patients of limited means to moderate the high price of his medicine or to give a pre-

scription for it, so I had a bottle of it analyzed by my colleague *Hoppe-Seyler*. The analysis showed that the blue mixture consisted of a solution of bromide of potash (ʒ jss—ʒ vj) colored with indigo. Both patients had taken the remedy in considerable doses. At first only two tablespoonfuls were given daily—but after ten days four, after ten days more six spoonfuls; after that the dose was increased more slowly, being gradually raised to ten, fifteen, and twenty tablespoonfuls. So it appeared that in this case, as in most others where secret remedies prove useful, it was not the remedy but the mode of using it that was the secret; and I thought it probable that the contradictory assertions about the action of bromide of potash were greatly due to the fact that different observers had not given it with equal perseverance and in the same doses. I determined to imitate the treatment of the "specialist" where practicable, and induced other physicians to do the same—and I became convinced that, thus used, in large doses for a long time, although it will not cure all cases of epilepsy, it will in many cases relieve the attacks for a considerable period, and in some will even remove advanced impairment of the psychical functions. Neither in my own practice, nor in that of others, have I, of late, seen a case where the intervals between the attacks did not grow longer. Even patients who had previously received no benefit from the bromide of potash began to improve, when the dose was raised to eight or ten tablespoonfuls of the above solution daily. On long-continued administration of the bromide of potash, I have often observed a papulous exanthema, and in one case an extensive furunculosis which disappeared on stopping the medicine, and again recurred after it had been resumed. After taking large doses of the bromide for a time, some patients complained of loss of appetite, confusion in the head, and catarrh of the air-passages, just as they do after protracted use of preparations of iodine. Lastly, in two cases there was some psychical change, evinced by great restlessness, and diminished mental activity. In most cases, there were none of these unpleasant concomitants; hence I feel justified in urgently recommending further trials of the bromide of potash in epilepsy, especially in long-continued and large doses.

To these specifics may be added a large number of medicaments which have been recommended with more or less urgency. Among these are assafœtida, folia aurantiorum, radix pæonæ, viscum album, oleum terebinthina, and oleum animale Dippelii, indigo, and others. It is a good rule, in administering specifics, to give them exactly in the form and dose prescribed, and not to pass too soon from one article to another; and, on the other hand, to bear in mind the

fact that there are a great number of medicines which, for a while, seem to do good, but which afterward fail as though the system had become used to them.

The indications from the disease itself require that the patients should be protected from the injuries which they are apt to inflict upon themselves during the fits. Whenever circumstances permit, an epileptic should not be permitted to go unwatched. This is the best and surest preventive. It is a good plan to let the patient sleep in a bed made with high sides, like a child's crib, but such measures do not dispense with the necessity of further watching, since, if the patient were to happen to lie upon his face in such a bed, he might readily be smothered. The patient must not be bound nor held fast during the fit, nor should his thumbs be forcibly unclinched. Many people believe that the attack will soon subside if this be done. After the fit is over, those patients generally feel best who have been allowed to struggle through it unmolested. The next symptomatic indication is to take the measures already recommended for the purpose of averting a coming paroxysm and of cutting it short. On the whole, compression of a limb, from which the aura seems to proceed, is not advisable, even although we may avert the fit by so doing, since, in the first place, the patient feels worse after thus repressing an attack than if he had had one; and, in the second, because his next seizure is apt to be of unusual violence. This is also true as regards the use of strong emetics at the commencement of the aura. Sometimes it is possible to arrest the fit by compression of the carotids. Such a practice, however, is very difficult to carry out during the attack, and might do harm if awkwardly performed; hence we would hardly recommend it.[1]

CHAPTER IV.

ECLAMPSIA INFANTUM.

The eclampsia of parturient and of puerperal women is very often dependent upon a morbid condition of the gravid uterus, upon the retention of portions of placenta after delivery, or upon other and unknown disorders of pregnancy or of child-bed. We therefore refer the subjects of puerperal and parturient eclampsia to the text-books of obstetrics, just as we have already referred those of other puerperal conditions of the womb, ovaries, and vagina.

Etiology.—We have called eclampsia an acute epilepsy. Here, too, there are convulsions with loss of consciousness; but, unlike epilepsy, the recurrence of the fit, instead of continuing for years, lasts

for a few days or hours only, at the end of which time the eclampsia terminates either in recovery or death.

As regards the pathogeny of eclampsia, the same remarks apply which we have already made concerning epilepsy. For reasons already given in detail, we may regard it as proved that the morbid irritability of the motor nerves, of which the convulsions are an indication, originates from the medulla oblongata and base of the brain. There also seems to be no doubt but that causes, similar to those which result in an habitual irritative condition of the medulla oblongata, and epilepsy, may often (and especially during childhood) induce an acute temporary state of irritability in the medulla with eclampsia. Strictly speaking, the animals upon which *Tenner* and *Küssmaul* experimented died of eclampsia, and not of epilepsy. It is more than probable, also, that congestion may be capable of inducing a state of acute irritability of the medulla, and may thus give rise to eclamptic seizures. This sometimes happens, owing to the introduction of foreign matter into the blood, as is shown in the rapidly fatal convulsions which sometimes arise in uræmia and in cases of narcotic poisoning. In this category, too, the convulsions belong, which arise—especially in children—in consequence of infection from miasmatic or contagious diseases, as also are those which usher in an attack of scarlatina, measles, or small-pox; as well as those arising from the febrile crasis, and from fever-heat, and which often announce the commencement of pneumonia and other inflammatory diseases of childhood. The convulsions proceeding from the progress of acute diseases of the brain and spinal marrow, and which are analogous to the form of epilepsy arising from cerebral tumors and other chronic disorders of the brain, are not counted as eclampsia, or, if so counted, are distinguished as a separate and symptomatic form. Convulsions, however, which proceed from excitement of the cerebral ganglia, transmitted thence to the medulla oblongata, and which are the result of terror and other emotions, are regarded as true eclampsia. Most frequently, however, the medulla oblongata seems to derive the morbidly irritative state, from which eclampsia proceeds, from an excitement which is transmitted to it from the peripheral nerves. To this class belong the convulsions from teething and from intestinal worms, and those which occur from painful injury of the skin.

The antagonism between the hemispheres and the basilar portion of the brain is as great in this disorder as it is in epilepsy; and we are quite unable to explain why the convulsions are accompanied by loss of consciousness.

With regard to its etiology, it is to be remarked that, at the period during which a state of habitual irritation of the medulla is most rare,

its predisposition to acute irritation is greatest. Newly-born infants, and those who are but a few months old, are the most frequent sufferers from eclampsia. The disease is rare after the first dentition, and still more rare after the second. The tendency to eclampsia is often congenital, all the children of a family sometimes being afflicted in this way. We do not know what the diseases or constitutional defects are, on the part of the parents, which determine this predisposition in their children. It occurs in vigorous, full-blooded children, as well as in the puny and anæmic, and in boys quite as often as in girls, perhaps oftener. Besides the exciting cause of the disease already mentioned, the practice of allowing the child to nurse immediately after the mother has been extremely angry, is believed to be a frequent cause of eclampsia. Inexplicable as such an idea may be, it would be neither judicious nor right to slight it, and to oppose the appropriate precautionary measures, which consist in not letting the child have the breast immediately after the mother has been angry, until after the breast has been drawn.

Symptoms and Course.—The only form of the disease which is apt to set in suddenly and without warning is that which, during childhood, often substitutes the chill of older persons as a precursory symptom of the acute exanthemata in pneumonia, and in other inflammatory disorders. For a day or two the child is restless during sleep, and sleeps with its eyes partly open, contorting its face and grating its teeth from time to time, and starting when touched. Even while awake, a difference in its manner is observable. The child is cross, does not seem to enjoy its play, cries a great deal, and often changes color. The character of an eclamptic fit is precisely like that of an epileptic one. Here, too, at first, there are usually tonic convulsions, which last for some moments; the head is thrown back, the extremities extended, the eyes turned up, and the respiratory movements arrested. Then, clonic spasms begin, which extend over the muscles of the face, head, trunk, and extremities, throwing the entire body (or, what is more rare, one side of it alone) into convulsive motion. The spasms equal the epileptic convulsions in violence. The face is reddened and slightly cyanotic; frothy saliva appears upon the lips; the skin is bathed in sweat; the belly is inflated from air which has been swallowed; the respiration is much embarrassed, and the pulse is small and frequent. At the same time, consciousness is entirely suspended, and with it all sensation, even of the most powerful stimulus, is extinguished. A fit of this kind, however, rarely passes off as soon as an epileptic fit, but often lasts from a quarter to half an hour, or even longer. In private practice, the physician far more frequently has opportunity to see eclamptic fits than epileptic ones, as the former

often last until he arrives, while the latter usually pass over while the messenger is still seeking him. A year or two ago I saw a child in an eclamptic fit, which lasted for twenty-four hours, without interruption, although there were occasional remissions. The fit usually terminates with a long-drawn deep expiration, often, too, by a profuse evacuation from the bowels. This rarely occurs suddenly during the height of the attack, but more usually after its violence has somewhat subsided. The child then falls into a deep sleep, and if, next day, we do not make our visit too early, we may find it playing busily, as if nothing had occurred. But it frequently happens that there is more than one fit. A series of them often succeed one another at short intervals. A repetition of them may be anticipated, when the sleep is not sound after the first one has subsided, and when the children throw themselves about, gnash their teeth, and when their limbs twitch from time to time. The subsequent fits resemble the first one in all essential points, differing only in their degree of violence and in their duration. Many children never have more than one eclamptic attack. In others, they recur from time to time. The more plainly it can be shown that these recurrences are the result of fresh irritation, so much the less doubt will there be that we have an eclampsia to treat, and not epilepsy. And, on the other hand, the less distinctly demonstrable the exciting cause of the repeated paroxysms is, so much the more doubt will there be whether they are eclamptic or epileptic. This point can never be determined at the first fit, since its exciting cause often enough eludes detection. Death may occur, during the fit, from arrest of the respiration, and acute carbonic-acid poisoning, from exhaustion, or during the subsequent period of coma. Eclampsia is a very dangerous complaint among children during the first months of life, and a large proportion of those attacked die. In older children the prognosis is more favorable, and the disease usually terminates in recovery. It will be readily understood that both children and older persons are apt to die where the convulsions, with loss of consciousness, are an accompaniment of grave disease of the brain or spinal marrow, although in such instances it can hardly be said that they die of eclampsia. The same may be said of the so-called sequelæ of eclampsia. When seizures of this nature are followed by idiocy, palsy, squinting, or other grave disorder, it certainly is more than probable that they, as well as the fits, depend for their origin upon nutritive disorder of the central organ.

Treatment.—I believe that it is very difficult, and often quite impossible, to determine whether an attack of convulsions, accompanied by loss of consciousness, depends upon anæmia or hyperæmia of the brain, or whether either of these conditions exists. If the previous health of

the child have been good, and his appearance still be one of robustness and vigor during the fit, I would advise the administration of a clyster of one part vinegar and three parts water, the application of cold compresses to the head; and, if the convulsions do not soon subside, the application of leeches behind the ears, in numbers corresponding to the age of the patient. We cannot give medicine during the fit. After it is over, and should we fear a recurrence, we may give a laxative of calomel and jalap, or one or two doses of calomel with the oxide of zinc. On the other hand, if the child be puny and enfeebled by long sickness, let him have an enema of valerian, or camomile tea, with one or two drops of tincture of castor; or, if this be ineffectual, a clyster of emulsion of assafœtida (℈ss—j to ℥ iv). Sinapisms may also be applied to his calves, and he may be put into a warm bath. Other means are not to be had recourse to until after the fit has subsided. Apart from the cases in which eclampsia is symptomatic of cerebral disease, of uræmia, or the initiatory stage of some acute disorder, it is of importance to determine from which province of the nervous system the morbid irritation proceeds which is acting upon the medulla oblongata, for it will depend upon such determination whether we give a laxative, an emetic, an antacid, or an anthelmintic, or whether we ought to resort to some other course of treatment. I consider it unjustifiable to dose every child, who has had an eclamptic fit, with calomel and oxide of zinc, "in order to prevent his having another." If the stupor which follows the paroxysm be very profound, cold affusion should be prescribed. On the other hand, if there be severe collapse, we should order wine, camphor, and musk, and other stimulants.

CHAPTER V.

HYSTERIA.

Etiology.—Hysteria is even still less susceptible of classification in any particular category of nervous diseases than are epilepsy and eclampsia. In this protean malady, derangement of the sensory, motor, and psychical functions is nearly always combined with disorder of the circulatory and nutritive systems. Sometimes one set of symptoms predominates, sometimes another; and there often is an exalted excitability in one part of the nervous system, expressed in the form of hyperæsthesia or spasm, and which is complicated with interrupted irritability of some other region, evinced by anæsthesia and paralysis. We cannot as yet give a satisfactory answer to the question as to the existence of actual although impalpable material alteration in the nervous elements, to which the various forms of nervous derangement characteris-

tic of hysteria are ascribable. For instance, although most patients are extremely susceptible to external influences, yet this may be due quite as well to exalted irritability of the peripheral nerves as to an over-excitability of those portions of the brain whence consciousness of the impression is derived. The former supposition is contradicted by the wide-spread character of the hyperæsthesia, as well as by the simultaneous derangement of the psychical function; the latter by the intensity of the reflex action by which the hyperæsthesia is accompanied. The latter condition is only ascribable to increased irritability of the peripheral nerves, or to coexistent increase of irritability in the spinal ganglia. Hence the most plausible theory of the origin of hysteria is that to which *Hasse* adheres, namely, that the affection springs from a nutritive derangement of the general nervous system, both central and peripheral.

The facts that hysteria is observed almost exclusively in females, and principally in females between the age of puberty and that of the extinction of the sexual function, and that in a great number of cases hysteria is accompanied by a morbid condition of the sexual organs, have given rise to the supposition that hysteria is a disorder of the general nervous system originating in the nerves of the organs of generation. This theory, although somewhat too narrow, is true in a large number of cases. We have come to the conclusion, from the various phenomena described in previous chapters, that disease not unfrequently is transmitted from the nerves which it immediately affects to other nerves, and to central organs; and by analogy we may infer that a morbid state of the nerves of the sexual apparatus may extend to those of the rest of the body, as well as to the central organs. The mild derangements of innervation, the hyperæsthesia, the augmented reflex irritability, and the psychical disorder which shows itself in some women during the period of menstruation, would seem to depend upon a process of this nature, and, as it were, form a physiological analogue to the pathological state of the sexual organs which exists in hysteria. There cannot be any doubt that some cases of hysteria arise in this manner. If a uterine infarction should develop after an abortion or a severe labor in a woman who has previously been healthy, and should this be accompanied by a well-marked hysteria; should the hysteria continue as long as the uterine disease lasts, and should it disappear as soon as we succeed in discussing the infarction, it would be manifest that to the uterus alone were due the manifold nervous derangements which we call hysteria. All diseases of the womb and ovaries do not exert an equal influence in producing this disease. Besides the infarctions, it is more especially the ulcerations of the os uteri and the flexions of the womb which induce hys-

terical symptoms, while, in cases of malignant growth and in destructive affections, hysteria is much less common. Of the ovarian diseases, the dermoid cysts of moderate size cause hysteria much oftener than do the very large sacs resulting from cystoid growth. In some cases irritation of the genitals arising from excessive coitus, or imperfectly effected coitus, from onanism, or from simple sexual excitement, has an influence upon the nervous system, similar to that of the textural lesions of the sexual apparatus above mentioned. But it would be both narrow-minded and frivolous, and indicative of a most imperfect comprehension of the nature of woman, to ascribe all cases of hysteria, whose source could not be traced to structural change of the genitals, to over-excited sexual appetite, or to its unnatural gratification. I certainly am no optimist, and indeed rather incline in the other direction, but I cannot believe that all the hysterical widows and old maids who are hysterical, without exhibiting any structural disease of their genital apparatus, suffer from suppressed sexual passion, or gratify it in an illegitimate manner.

Where there is much predisposition to hysteria, it may proceed from any other organ of the body which may happen to be diseased. I have seen a strongly-pronounced case of it in a young girl with a tedious affection of the stomach, but whose sexual function was perfectly normal.

We must entirely agree with *Hasse*, in his delicate and excellent description of hysteria, that the frequence with which hysteria occurs in childless women, in widows, and in old maids of the upper class of society, is attributable rather to psychical than to physical influences. The effect of strong psychical impressions upon the nervous system at large is often quite evident even in perfectly-healthy subjects. Persons under the influence of great terror stand as if thunderstruck, unable to move from the spot; an angry man clinches his fists, bites his lips, and moves restlessly to and fro without act of his will. Moreover, we not unfrequently find that the influence of extreme terror or anguish of mind produces complete anæsthesia, and excessive intellectual exertion often gives rise to hyperæsthesia. Indeed, every day we have the opportunity of observing that mental emotion has an influence upon the excitability of the vaso-motor and nutritive nerves; that it causes the cheeks to redden or to grow pale, the muscles or the skin to contract or to relax, the tears or saliva to flow. If all these various disturbances of innervation can arise from transitory psychical impressions, it is easy to imagine that permanent nervous derangement, with disease of texture of the nervous system, may develop under the influence of the permanent mental emotions, which beset a woman who finds all the expectations and hopes of her life dis-

appointed, who believes that she has failed in her vocation, and who, under the depression consequent upon such feelings, is unable to divert her thoughts into other channels. I admit that the manner in which the continuous action of psychical impressions modifies the nutrition of the general nervous system, and thus leads to hysteria, is obscure; but it cannot be maintained that our insight into the mode in which disease of the genital nerves extends to other portions of the nervous system is any more clear. The state of mind which leads to hysteria depends not only upon external accidents, but much more upon the mental impression made by the accident upon the individual. A fate which may befall one person, without producing any apparent effect, may be the source of the deepest and most lasting depression in another. I heartily agree with *Hasse*, that, though hysteria is often seen in women wedded to impotent men, yet it originates quite frequently in the sombre feeling and miserable consciousness of a wasted life, which result when social claims of married life are not duly respected, or when the sentimental anticipations and fantastic ideals of a foolish girl are not realized.

The nutritive derangement of the nervous system from which hysteria arises may also be induced by improper nourishment. We have sufficient proof of this fact in the frequent occurrence of this complaint in cases of chlorosis and impoverishment of the blood, unaccompanied by any disease of the sexual organs, by sensual excitement or onanism, and where it cannot be traced in the remotest degree to the psychical impressions above alluded to.

There is great variety as to the degree of predisposition to hysteria. All the women with uterine infarction, or uterine flexion, or who have ulceration of the os uteri, are not necessarily hysterical, nor are all the old maids who believe their lives to have been thrown away, nor all the chlorotic girls. On the other hand, I have no hesitation in asserting that a tendency, either congenital or acquired, plays a much more important rôle in inducing this affection than all other causes mentioned hitherto. The truth of this assertion is easy of proof. If we examine large numbers of women, we find moderate degrees of uterine infarction, slight flexions of the uterus, and erosions of the os, to be so very common that the number of hysterical women would be equal to that of the non-hysterical ones, if affections like these alone sufficed to occasion hysteria, without the coexistence of a decided predisposition to such disease. It rarely begins to manifest itself before the twelfth or fifteenth year of life, and very seldom appears in old age. It frequently outlasts the period of child-bearing, and continues in a moderate degree during the climacteric years. The tendency to it is often congenital; but, although a patient may have

descended from an hysterical mother, and she, too, be descended from hysterical parents, this alone is no absolute proof of the existence of such hereditary tendency, as it may have proceeded from injudicious training, which has likewise propagated itself in the family for gen erations.

Constitution and temperament have no distinct effect upon the tendency to hysteria. On the other hand, the mode of life of a patient, and her education, have a most decided influence in this respect. The less a child is taught to control itself, the more it is allowed to indulge in immoderate grief over a broken toy; the more the rod is spared, when it gives way to outbursts of excessive anger or passion, stamping its feet and throwing itself upon the floor, all on account of some disappointed expectation, or the refusal of some request, so much the more apt will it afterward be to become hysterical. If we teach a child to be industrious, to be conscientious, and to control itself; if we prevent growing girls from knitting or doing worsted work all day long, or from occupying themselves in other ways, which permit of their indulging in dreams and reveries; if we keep improper books, likely to give them stilted ideas, out of their hands, we shall have done our best toward averting the danger of hysteria. Hysteria is a very rare phenomenon among men. Here, too, it usually proceeds from derangement of the sexual apparatus, although this is not always the case; but in men, likewise, it is only in instances of decided congenital or hereditary tendency to the disease that it can be excited by venereal excess, onanism, spermatorrhœa, and the like.

Symptoms and Course.—Owing to the complicated nature of the symptoms of hysteria, and to the varied character of its course, it is impossible to give a brief and comprehensive description of this disease. Hence we depart from our usual practice in describing hysteria, and, instead of drawing a picture of the affection itself, shall give a classified discussion of its symptoms.

Derangement of sensibility is a very common symptom, and is scarcely ever absent in any case of hysteria. *General hyperæsthesia*, or "nervousness," as the laity call it, is the first symptom of this class to which we shall refer. It often exists for years uncomplicated by any other form of the disease. This hyperæsthesia is sometimes evinced by an unusual acuteness of the senses. Some patients are able, by touch alone, to perceive the most trifling differences of weight and temperature, and thus to distinguish objects from each other, with their eyes shut, which healthy persons could not have distinguished. It is easy to understand that a faculty of this kind seems wonderful to the masses, and that it often is used for purposes of imposture. In a similar manner some patients have their sense

of smell developed to a degree usually found in brutes alone. They are able to distinguish persons and things in the most remarkable manner merely by their olfactory sense; or they may be able to hear and to recognize the step of a person who is still far off. Similar acuteness of the sense of taste has also been reported. Fortunately, however, hysterical hyperæsthesia very rarely takes the form of abnormal acuteness of the senses, or else the number of clairvoyants would be still greater than it is. Far more frequently the hyperæsthesia of hysteria shows itself in the annoyance produced by a very slight degree of stimulation of the nerves of special sense. While, in a healthy person, it requires a very loud sound, a very strong smell, a very bitter or acrid substance, or a very bright light, to produce an unpleasant effect upon the senses, hysterical persons complain if we elevate our voice a little in speaking, and beg us to talk in a whisper. They will often banish all flowers from their room because they cannot endure their odor; or will reject their food if it contain the slightest particle of spice. To some hysterical persons bright daylight is quite intolerable, so that they constantly keep their eyes closed; to others a red color is unendurable, and no one must approach them without first putting off any red ribbon or article of clothing they may be wearing. This great sensitiveness to comparatively slight stimulus is often associated with *idiosyncrasies*. Certain forms of irritation, which, from their quality rather than from their intensity, are repulsive to healthy subjects, afford a sense of gratification to hysterical persons, and conversely stimulants, which are pleasant to a well person, often offend the senses of one who is hysterical. It is notorious that many hysterical people love the smell of burnt feathers, and take assafœtida without repugnance, while the odor of hyacinths, violets, and other most agreeable perfumes is insupportable, to them.

Besides these signs of morbid irritability, there are other states of excitement of the sensory nerves which must be regarded as of a different character. In the first place, there are neuralgias, especially prosopalgia, migraine, mastodynia, and ischias, all of which are of common occurrence in hysteria. Closely related to these there is a form of pain which is also very common, and which is generally confined to one small point in the head (usually to one side of the sagittal suture), and which is known by the name of *clavus hystericus*. Besides this, there is the almost never-failing tenderness, on pressure, of the back, and finally those very peculiar hysterical affections of the joints (*Arthropathia hysterica*) which consist in pain in the articulation, often of such exquisite severity and so obstinate a duration as to be liable to be mistaken for grave inflammation. Morbid excitement of the nerves of special sense may also occur, which cannot be accounted

for by the action of any corresponding irritant. Some patients are never free from the sense of a certain taste or smell; some complain of roaring and buzzing in the ears, or of spots before their eyes. It is very remarkable that, besides these phenomena, indicative of exalted excitability, and of a morbid irritability of the sensory nerves, there should also be an anæsthesia, involving a variable extent of the surface of the body. It is doubtful whether this anæsthesia be attributable to suspension of function of the peripheral nerves, or to an extinction of irritability at the nerve centres. I consider it very hard to determine whether an hysterical person really is suffering from anæsthesia, or merely indulging the caprice of not showing signs of pain when pricked, pinched, or burnt in particular parts of the body. There is not the least doubt that such notions are of daily occurrence among hysterical patients. If they only knew what a puzzling and interesting subject this matter of anæsthesia is, the number of hysterical cases of this kind, no doubt, would increase vastly. I have seen a patient who did not move a muscle while two streaks were being burnt along her back with a red-hot iron, and yet there was not the slightest ground for believing that she had anæsthesia of the back.

All the derangements of sensation hitherto described have depended upon morbid irritability of the cutaneous nerves, and of the nerves of special sense. Connected with these there is a series of perverted sensations in internal organs. While, under ordinary circumstances, we have either no perception at all (or, at most, a very obscure one) of the condition of the internal viscera, as long as they are in good order, and while, without laying our hand upon the heart, we are unaware of its pulsation; and while respiration goes on without our consciousness of the necessity for such an act; and while we have no perception whatever of the ordinary state of our stomachs and bowels, yet hysterical individuals have the greatest variety of complaints to make as to the condition of their internal organs, and claim to suffer the most extraordinary sensations. They nearly all complain of palpitation of the heart—many of them of pulsation of the vessels. Upon examining the heart-beat and the pulse, we may easily satisfy ourselves that such sensations are but subjective ones; and that the shock of the heart is not really increased, nor is the pulse full and hard. It is the same with the shortness of breath. Sometimes the patients breathe laboriously and quickly, complain of the utmost oppression; but, after exclusion of the possible existence of any disease of the air-passages or circulation, or of a morbid state of the blood or nutrition, capable of accounting for such dyspnœa, we may assure ourselves that it is purely a case of hyperæsthesia or of perverted sensation. Besides this, although their digestion may be excellent, nearly

all hysterical patients complain of a sense of pressure and fulness in the region of the stomach, or else of cardialgia, and, independently of the colic which sometimes troubles them, give most extraordinary accounts of the sensations which they feel in their abdomens. Under this head also come the thirst, often observed in hysterical persons, and the frequently-recurring desire to empty the bladder, although it is not full. Perverted sensation in the sexual organs is much more rare than might be supposed, more so, indeed, than has been represented by many authorities. Husbands of hysterical women often declare that their wives evince a disinclination to sexual intercourse, and do not often exhibit much excitability. They seldom give an account of an opposite condition. Even in hysterical prostitutes it is exceptional to see symptoms of nymphomania. In other cases, again, coitus is very painful to the patient, although there may be no palpable lesion of the genital organs.

The derangements of the motor function observed in hysteria are scarcely less numerous and varied than those of sensation. Their most common form is that of hysterical convulsions. There is no doubt but that the morbid excitement of the motor nerves, which gives rise to hysterical spasms, proceeds from the spinal marrow and medulla oblongata. One of their characteristic signs is, that they never cause loss of consciousness. It is usually supposed that hysterical convulsions are of reflex origin, so that the spinal cord can only transmit the impressions conveyed to it from the sensory nerves to the motor nerves. As the convulsions frequently spring from impressions produced upon the sensory nerves, or the nerves of special sense, it seems probable that this theory is the correct one; hence, in cases where the convulsions seem to occur spontaneously, we must suppose that the cause has eluded our observation. In some cases, hysterical fits consist in a mere twitching of one or more of the limbs, especially of the arms. They often recur, at short intervals, for a while, whenever the temper of the patient is excited, and whenever stimuli, even of moderate strength, act upon the nerves of sensation or those of special sense. In other cases the spasms extend more or less over the entire body. They occur in violent paroxysms, and may almost assume the appearance of tetanic spasms, and still oftener of epileptic convulsions. Opisthotonos, pleurosthotonos, and orthotonos, are seen often enough in hysteria, as are also clonic convulsions, which set the face, trunk, and extremities into spasmodic motion. The patient often foams at the mouth, his thumbs are clinched in the palms of the hands—the only sign, by which the attack may be distinguished from an epileptic one, being loss of consciousness. Very often spasm involves the group of muscles which operate in producing some complex

act, such as yawning, laughing, crying, although the mental emotion by which they are generally induced is not present. In this way fits of spasmodic yawning, laughing, and weeping arise. Thus, spasmodic expiratory movements, combined with spasmodic contraction of the rima glottidis, and tension of the vocal cords, give rise to that obstinate cough known as hysteric cough, and to its modifications, consisting of barking and howling sounds. Spasmodic contraction of the œsophagus, extending from below upward, gives the patient a feeling as if a ball were rising from the epigastrium into the throat, a symptom commonly called *globus hystericus*. Very often hysterical persons are troubled for a quarter of an hour, or even for hours at a time, with eructations, which recur at short intervals, and during which a quantity of inodorous air is belched up, usually with a loud sound. If we watch the patient carefully, we can see, by the movements of her mouth and throat, that she has previously swallowed this air. As however, the majority of healthy persons are unaware of it, when they swallow air in the movements of chewing and deglutition which they make under the influence of violent nausea, I do not suppose that hysterical persons do it consciously and intentionally; hence I have classed this symptom among the disorders of motion. Just as anæsthesia and hyperæsthesia of the sensory nerves may coexist, so in the motor system hysterical convulsions may be accompanied by hysterical palsy. Sometimes a single extremity alone is involved; sometimes there is hemiplegia. The fact, that the electric contractility is preserved in the muscles of the palsied part, argues strongly against the peripheral origin of this form of paralysis. If the peripheral nerves were diseased, if they had lost their functional excitability, then electricity would be just as powerless to excite them as the will is. Now, in hysteric palsy, as every muscle which the patient is unable to contract voluntarily may be made to contract by application of the electrode to its nerves, the palsy must be of central origin. Moreover, the rapid changes which occur in hysterical palsy, especially the sudden way in which it sometimes subsides, proves that its source is some slight and easily-adjusted derangement of nutrition, and that it does not consist in serious structural change at the centre of volition, but, in some cases, an excessive indecision and incapacity on the part of the patient to make up her mind to move the limb seem to be the reasons for the palsy. I have no doubt that any one, as long as he is firmly persuaded that he is unable to perform a certain act, is indeed incapable of generating the necessary motor impulse. As the palsy in this class of cases is due to a perverted mental impression, it should, properly speaking, take rank among the psychical disturbances. Some time ago I saw a patient suffering from a hemiplegia, which had lasted for months. The

history of the case was, that, for years past, the patient had suffered from a similar palsy, which had disappeared at times, and then had returned again. This peculiar behavior, together with other hysterical symptoms, left no doubt as to the hysterical nature of the case. The patient had repeatedly been assured that she could be cured by the application of electricity, and, as her reception at the clinic was delayed, her anticipations as to the success of the treatment were wrought up to the utmost pitch. The opening of her firmly-clinched hands, upon the first application of the electrodes, unmistakably made a great impression upon the patient, and from that time forth the paralysis improved, and disappeared altogether in the course of a few weeks. No doubt, any other remedy, in which the patient had felt equal confidence, would have been quite as successful.[2]

Among the derangements of the vaso-motor and nutritive nervous systems, the most striking are the uneven and fluctuating state of the circulation in peripheral regions. Most patients constantly have cold hands and feet, while, without apparent cause, the natural color of the face gives place to a glowing redness, often accompanied by a disagreeable burning sensation. It is uncertain whether the secretion of saliva, and that of the juices of the stomach and intestines, are also modified by spasmodic contraction or paralytic dilatation of the vessels of these organs; on the other hand, it undoubtedly is through derangement of innervation that the determination of blood to the kidneys arises, which causes the profuse secretion of urine so often seen in hysteria. This urine, which is voided in large quantities, contains but little of the solid constituents of urine, is of a limpid appearance, and is often described as hysterical urine—*urina spastica.*

It is very difficult to furnish a brief and comprehensive description of the psychical derangements observed in hysteria. In the first place, at the outset of the affection, we are struck by the rapid fluctuations which take place in the spirits of the patient, and by the sudden transitions from the most unbounded gayety to the profoundest gloom. These symptoms are in part ascribable to the physical hyperæsthesia described above, and in part to psychical hyperæsthesia, by which it is accompanied. As mental impressions produce an unusual influence upon the temper of the patients, so, too, suggestions which would produce no apparent effect upon the spirits of a healthy person produce a sense of annoyance or of repugnance in an hysterical one, although more rarely the sensation is one of gratification and pleasure. While the affection is still recent, it is almost always possible, by adroitly conducting the conversation, in the course of a few minutes, to make the patient laugh and weep alternately. It would seem also that, besides this psychical hyperæsthesia, there may also be a psychical

idiosyncrasy in hysteria, and that the odd and often very extraordinary state of mind into which the patient falls is to be ascribed to this. As, however, the real and imaginary impressions which inspire the patient with a sense of disgust or displeasure are the predominant ones, her spirits gradually become more and more depressed. She is constantly sad, unhappy, and in despair about her fate, even although she may be in possession of every thing which can tend to make life enjoyable. This constant and apparently unreasonable lamentation and weeping gradually tire out the sympathy of the friends of the patient. Her relatives become indifferent to her troubles, do not listen to her complainings, or allow it to be perceived that they are becoming tired of them. Unfortunately, hysterical persons often become objects of ridicule to inexperienced physicians. It is to this absence of sympathy and to this gradual diminution of interest that we may most reasonably ascribe the tendency which develops in nearly all hysterical patients to exaggerate their complaints and to feign diseases—a tendency which, although really a symptom of the malady, does away with the last vestige of sympathy for their condition. Cases are on record where patients have undergone the most painful operations, in order to regain the attention and sympathy which have been withheld from them. *Krukenberg* tells of an hysterical girl, at his clinic, who maltreated a wound upon her skin with irritating substances until amputation became necessary, and who, after the stump began to heal, recommenced the practice. The capacity of such a patient for inventing conditions calculated to excite notice or sympathy is something incredible. It is often very difficult to separate the truth from falsehood. Credulous persons are often duped, and we should make it a rule to accept all unusual reports with the utmost distrust, such as, that the patient never takes any food, that she never passes either water or fæces, that she has vomited blood or maggots, or other odd objects. It is a very common occurrence for a patient to declare that she cannot make water, and for her to submit to the passage of a catheter twice a day. It is equally common for her to remain in bed for months or years, asserting that she cannot stand upright. It is easy to see what a treasure animal magnetism must be to hysterical persons, and with what alacrity they submit to the manipulations of the "magnetizers," and that, having once "got into magnetic rapport" with some other person, and thus become enabled to perform all manner of new tricks, they leave off their old ones, and thus are "cured of the most wonderful diseases by animal magnetism." In my opinion, it is only necessary for the right man to appear (like the magic tailor in *Immermann's* "Münch-hausen"), in order to convert any decidedly hysterical female into a "somnambulist" or clairvoyant. At the same

time it is to be admitted that fanatical enthusiasts, who devoutly believe in their own wonderful gifts, and who are sustained in such belief by their experiments with hysterical persons, are capable of much more than mere impostors, who speculate in animal magnetism. The power of reason usually remains unimpaired in hysteria. Like other people, they are able to connect one idea with another, and to form correct conclusions, although they are so preoccupied with a sense of their own sufferings as to be unwilling to think of other matters. A very prominent peculiarity in hysteria consists in the loss of power of the will to control the movements of the body. This peculiarity is also attributable to the almost absolute control which the mental excitement has obtained. Even persons in good health, when agitated in mind, pay less attention to the motions of their limbs, and do not, by any action of their will, restrain their reflex movements which arise during the agitation. In spite of the authority of *Romberg*, I cannot agree with those who believe the enfeeblement of will of the hysterical to be the result of a reflex action of such intensity as to overcome the action of volition. The real state of the case I believe to be exactly the reverse. At my clinic, I have nearly always succeeded in producing a fit of hysterics in suitable cases, or in exciting a slight attack into one of great violence, by expressing great sympathy with the patient, and by assuring her that the fit was going to be a bad one, thereby so agitating her as to hinder her from bringing her will to act upon her motions. On the other hand, by treating the patient roughly during the paroxysm, by throwing glass after glass of water in her face, and by threatening to keep it up to the end of the attack, I have nearly always been able to stimulate the patient to a vigorous exertion of her will, and thereby have put an end to the involuntary movements. Uneducated bystanders, and superficial observers, when they see how manifestly the fits depend upon psychical influences, are very apt unjustly to suspect the patient of an imposture. Besides, the course of the attack, as described above, corresponds exactly with the well-established facts of physiology regarding reflex action and the effect of the will upon it.

There is much variety as to the course, duration, and results of hysteria. In most instances the disease comes on gradually. At first, the only symptoms are those of bodily and mental hyperæsthesia and their consequences; and it is not until afterward that the convulsions and other symptoms of greater or less violence show themselves. Sometimes they never appear. In rare instances the complaint begins with an attack of hysterics running an acute course, the other symptoms not making their appearance until afterward. At the menstrual period, and immediately prior to it, the disease is almost

always aggravated; sometimes, indeed, there never are any fits, excepting at this period. There is no fixed rule as to the duration of hysteria. It may continue for years, with varying intensity, although, during the climacteric years, it nearly always becomes milder. Recovery is not uncommon, the physician's art having many a triumph over hysteria to celebrate. There are plenty of cases, it is true, which are never cured, and, indeed, which do not even improve. Sometimes the malady runs into epilepsy or insanity. Death from hysteria, however, is rare. There are only a few scattered cases on record, in which death has occurred during violent convulsions, probably owing to embarrassment of respiration.

Treatment.—After what has been said above regarding the effect of education and of habit in inducing hysteria, the necessary prophylactic measures so important to the patient will have become sufficiently evident, and will not require any further specification.

Cases where there is no doubt that the nervous derangement proceeds from disease of the sexual organs, call for appropriate treatment of the infarction, ulcer, flexion, or other disease of the womb which may be present. For further remarks upon this subject we refer to the second division of this volume. Where the hysteria is the result of psychical influences, and nevertheless the patient is compelled to submit to the extremely unpleasant procedures necessary for the application of leeches or caustic to the os uteri, the malady is almost always aggravated. In hospital practice, it generally is impossible to meet the indication as to cause in cases of this kind; but, in private practice, a physician, who is intrusted by his patients with a most intimate knowledge of their private relations, is often able to exert the most happy influence in this variety of hysteria. It is impossible to lay down any general rules of procedure. When the disease depends upon impoverishment of the blood and upon chlorosis, the indication as to cause requires that we should endeavor to improve the state of the blood by the exhibition of iron and an appropriate diet. By means of such treatment our object is soon effected, and the hysteria disappears as the red cheeks return, without it having been necessary for us to have recourse to antihysteric remedies. Hysteria which is traceable to anæmia is perhaps the most satisfactory of all forms of the disease to treat.

The indications as to the disease demand that we shall attempt to allay the nutritive derangement of the nervous system upon which the hysterical phenomena depend. We must not expect to effect a final cure of an hysteria by the mere healing of an erosion upon the os uteri, although this may have been the original cause of the malady. It may happen, but is by no means the rule. The proper means for attaining

our object are, first, remedies which have an active influence upon the general nutrition of the system. Secondly, those which seem to act specifically upon the nervous system—the so-called nervines. The success in the relief of hysteria, of which the hydropaths justly boast, is due to the effect of the so-called hydropathic treatment. When the cause has been removed without benefit to the patient, or if it be impracticable to allay the cause of the hysteria, the cold-water cure is, in many cases, strongly to be recommended. We must warn the patient from the very first, that the "cure" cannot have the desired effect in a few weeks, and that their abode at the water-cure establishment must be continued for several months at least. Nor is it advisable to allow a patient to undertake the water-cure at her own house, as it is of the utmost importance that the treatment should not be conducted by halves. The use of sea-baths also is often of remarkable benefit in hysteria. When the patient is vigorous and well-nourished, the springs of Marienbad, Kissingen, and Franzenbad, are often of great service. The action of these springs is to be ascribed to the modification which they exert upon nutrition. The nervines which have the greatest reputation as remedies against hysterics are castor, valerian, hartshorn, assafœtida, and other evil-smelling and ill-tasting articles. The greatest skeptic cannot deny that a cup of valerian tea, a few drops of the tincture of valerian, or a tincture of castor, taken by the mouth, or an injection of the infusion of valerian, or an emulsion of assafœtida, often act admirably as palliatives, although no radical relief can be effected by such articles. I have accidentally hit upon a nervine of great efficacy in hysteria, and have made use of it with signal effect in many cases where there was no indication for the local treatment of uterine disease, or else, where the hysteric symptoms persisted although the local uterine affection had been cured. I mean the chloride of sodium and gold. I had read that *Dr. Martini*, of Biberach, regarded this article as an efficient remedy against the various diseases of the womb and ovaries. As the diseases which this gentleman (a much-respected gynecologist in his district) claims to have cured by this drug, for the most part, belong to that class of disorders which are incapable of resolution, and which are incurable, in the strictest sense of the word, and as I had no reason to doubt the veracity of *Dr. Martini*, I could only infer that the cases had been imperfectly observed. My suspicion that the chloride of gold and sodium, like other metallic articles, was an active nervine, and that the improvement effected upon *Dr. Martini's* patients was probably due to this property, was fully confirmed by what followed. After having employed the medicine in question for several years, and in a great number of cases, and, encouraged by my success, having recommended

it to my pupils as one of the most effective of nervines, I ascertained that the chloride of gold has been recognized as a special re-agent upon the nerve-tissues, and that it is much used for this purpose in microscopic investigations. Such a discovery, probably, would never have induced me to make use of it as a remedy, but now this piece of information is of great importance to me, as a confirmation of the fact that the curative action of this remedy had been rightly interpreted. I prescribe the chloride of gold and sodium in the form of a pill (℞ auric. chlorat. natronat. gr. v; gummi tragacanth. 3 j; sacc. alb. q. s. u. f. pil. No. 40). Of these pills I at first order one to be taken an hour after dinner, and another an hour after supper. Afterward I order two pills to be taken at these hours, and gradually increase the dose up to eight pills daily.

In some cases of severe hysteria where fulfilling the causal indications does not answer the purpose, or where the origin of extensive hysterical disturbances of innervation cannot be discovered, either in the sexual apparatus or other organs, I have experienced some excellent results from the use of bromide of potash in increasing doses (fully described in the treatment of epilepsy), while in other cases it failed entirely. An esteemed colleague also, who has a large consultation practice, informs me that, when he is unable to refer severe nervous symptoms to structural changes in the central organs or the peripheral nerves, he often resorts to this effective but empirical remedy.

Moral treatment is of the utmost importance in all cases of hysteria, whatever may be the source of the disease. *Romberg* very properly observes: "Psychical treatment is of such importance that, without it, all other remedies fail." Every thing depends upon our exercising the patient to "oppose the impulse of the will to the reflex impulse." It of course depends upon the peculiarities of the patient as to what measures we shall resort to in special instances. When they have shown themselves to be willing and obedient, and seemed to place reliance upon my directions, I have ordered them to take a cold shower-bath twice a day, and to keep under the shower as long as the utmost exertion of their will would enable them to do so.

[Electricity is serviceable not only against certain symptoms of hysteria, such as paralysis, anæsthesia, neuralgia, etc., but it is asserted to be of positive benefit for the general disease. The best application is the induced current in the form of "general electrization," as recommended by *Beard* and *Rockwell*, where currents of moderate intensity are passed over the entire surface.]

CHAPTER VI.

CATALEPSY.

ETIOLOGY.—Catalepsy belongs to the neuroses of stability (*Stabilitäts-neurosen*), according to the classification of *Blazius*. During a cataleptic fit, the limbs of a patient remain in the attitude in which the patient voluntarily placed them prior to its commencement, or in which they may be placed by a bystander during the seizure. The limbs do not sink by reason of their weight, nor can the patient of his own will bring them into any other position. The resistance to gravitation opposed by the limbs proves that the muscles must be in a state more or less of contraction. In all conditions in which the muscles are fully relaxed, as in a swoon, or after death, the limbs fall of their own weight as soon as support is withdrawn from them. It would seem most natural to attribute the steady continuance of the limbs in one attitude to a continued excitement of the nerves which induce the muscular action requisite to produce such an attitude. This explanation, however, is contradicted by the other phenomenon, for, if we alter the position of the limbs of the patient, the new posture is retained, just as the former one was. It is contrary to all experience that a change of attitude in a limb, effected by foreign interference, should arrest the action of one nerve and induce action in others. Cataleptic attacks are not sufficiently common to enable us to decide these points positively; but it is most likely that all the motor nerves are in a state of medium excitement in this disease, and hence that *all* the muscles of the body are in a state of contraction sufficient to counteract the resistance afforded by the weight of the limbs. The facility with which the posture of a limb may be changed (*flexibilitas cerea*), and the fact that a limb remains bent, if we bend it, or straight, if we straighten it, lead to the conclusion that the contraction of the antagonistic muscles fully preserves their equipoise. The supposition that the medium state of irritability of the motor nerves, which is the cause of this condition, proceeds from the spinal marrow, is also the most satisfactory and general. The incapacity of the patients to modify this excited condition of the motor nerves and contractile state of the muscles by the force of their will indicates that there is also a morbid state of the brain. In cases of catalepsy, where consciousness is entirely suspended, no struggles occur; and, in cases where consciousness is retained, the patients wish to move, but cannot, because that particular part of the brain is thrown out of function,

whose duty it is to conduct the impressions from the central organs of imagination and volition to the motor nerves.

Catalepsy is quite common among the insane, especially among persons suffering from *melancholia attonita*. It also sometimes precedes the convulsive attacks of hysteria. Cataleptic fits may also accompany St. Vitus's dance, the *chorea Germanorum*, tarantism, and other epidemic and endemic forms of convulsion of psychical origin, and which *Romberg* aptly terms psychical convulsions. It is very rarely met with as an independent affection in persons whose health is otherwise good. Children and young persons seem to be most liable to its attacks. Mental emotion is said to be its principal exciting cause; indeed, in healthy individuals, slight indications of this morbid condition are sometimes observable as a result of such impressions. It is a very common thing to see persons, under the influence of sudden fear or horror, standing motionless, with outstretched hands, until the emotion subsides.

Symptoms and Course.—In my description of the symptoms and course of catalepsy, as an independent disease, I must rely entirely upon the representations of others, since all the cases which I have had an opportunity of observing personally have inspired me with the suspicion that they were simulated. The precursors of a cataleptic fit are said to be headache, dizziness, buzzing in the ears, broken sleep, extreme irritability, and other symptoms of nervous derangement. The paroxysm itself sets in suddenly. The patient remains motionless as a statue in the attitude in which he may happen to be at the moment of the attack. At first, some force is required to move the limbs; afterward, they can be moved with ease, and it is possible to adjust them in any desired posture, and they will retain that posture much longer than a healthy person would be able to retain it of his own will. During the fit, consciousness and sensibility to external impressions are either entirely suspended, or else, though the senses may be retained, and though external impressions are perceived, the patient is unable, either by word or act, to give any sign of consciousness. The respiratory movements and the beat of the heart and pulse are usually so feeble as scarcely to be perceptible. The urinary and alvine evacuations usually are arrested. Deglutition proceeds undisturbed, if the bolus be thrust far enough back into the pharynx. Generally speaking, such a fit only lasts a few minutes; more rarely it continues for several hours or several days. When it is over, the patients yawn and sigh like persons who have waked out of a profound sleep. If the seizure be a very transient one, and be accompanied by loss of consciousness, the patient often is quite unaware that any thing unusual has happened to him, and, after the fit is over

goes on with his business quite undisturbed, taking it up where he left it off. In other cases, the patient remains stunned and dizzy, for a while complaining of a feeling of confusion in the head. There is often but one attack; more rarely there is a series of them, recurring at varying intervals. Between the fits the health is good, unless there be complications. Recovery is the most frequent termination of simple catalepsy. The dread lest cataleptic patients should be buried alive has become exploded in modern days. It is stated that, in some cases of the disease, the length of the fits, and the frequence of their recurrence, and the consequent diminution in the supply of nourishment to the patient, have sometimes resulted in marasmus, and even in death. In such cases, however, it is probable that there was a complication of diseases.

Treatment.—In spite of the old caution, not to interfere too actively with a cataleptic fit, I should not hesitate to resort to affusion of cold water, to apply a strong electric current, and, unless the respiration and pulse should seem too feeble, to give an emetic. In a protracted seizure, it may become necessary to feed the patient through an œsophagus tube. As the treatment of the case between the fits must be addressed to their apparent cause, to any derangement in the nutrition of the patient, or to any other symptom which may appear, no fixed rules can be laid down for our guidance.

CHAPTER VII.

HYPOCHONDRIASIS.

Etiology.—Properly speaking, hypochondriasis is a disease of the mind, and, in the books on psychiatria, is usually classed with melancholy, lypemania, and phrenalgia, that is, to that class of diseases in which, as their names indicate, the mind of the individual is oppressed by a painful impression. The hypochondriac is always plagued by the idea that he is sick, or that he is going to be sick. Hence, in the opinion of *Guislaine*, an appropriate name for the disease would be *pathophobia* or *monopathophobia*. All persons who are possessed by the idea that they are sick, however, are not to be regarded as hypochondriacs, but only those in whom such an impression constitutes a symptom of disease. A father of a family, who is informed by his physician that he is suffering from some incurable malady, and from that hour is insane upon that point, suffering constant apprehension, and watching his bodily condition with all the attention and anxiety of a hypochondriac, quite assumes the aspect of one laboring under this disease. He is not a hypochondriac, however, for his

mental and bodily derangement correspond, and do not stand in contradiction to the psychical peculiarities of the patient prior to his discovery.

Like all symptoms of mental disease, hypochondriasis proceeds from nutritive derangement of the central organs of all psychical action, but we are not able to trace back the morbid state of mind, characteristic of this malady, to any particular lesion of the brain. We can scarcely ever point out the nutritive disorders of the brain to which the derangements of its functions are due in any other form of insanity, and, in hypochondriasis, it is equally impracticable to trace the causes of the morbid mental condition to cerebral lesion. Where a tendency to the disease exists, it may arise either from psychical or from physical influences. There is no objection to the application of the terms *hypochondria sine materia* to that form of the affection which proceeds from psychical causes, and *hypochondria cum materia* to that which arises from physical influences, but the expressions are not to be employed in any other sense.

The predisposition to hypochondriasis is very slight indeed during childhood, and is far less in females than in males. It is greatest between the ages of twenty and forty. Not unfrequently it is of congenital origin. In other instances it seems to proceed from debilitating influences, such as sexual excess, onanism, digestive disorder, or want of fresh air, as well as from an inactive mode of life, or immoderate self-indulgence, from disappointment, failure of speculations, and an ill-selected career.

The exciting causes of hypochondriasis are, first, physical diseases. Certain morbid conditions are more liable to cause hypochondriasis than others are, or (to speak more precisely) to produce those material lesions in the brain which are the cause of hypochondriasis. The principal of these are the gastric affections, especially chronic gastric or intestinal catarrh; next come diseases of the genitals, and, finally, gonorrhœa and syphilis. In the latter, however, the mental impression made by the disease ought to be taken into account quite as much as its physical effect. If these diseases sufficed of themselves to produce such disorders, the world would be full of hypochondriacs. Regarding them as mere exciting causes, however, only capable of bringing on the disease where a predisposition to it already exists, the disproportion between the frequence of gastric catarrh, of syphilis, or of clap, and that of hypochondriasis, will not appear at all remarkable.

The operation of mental impressions has a very similar effect. The most important of these consists in the reading of those pernicious books of "popular medicine," innumerable copies of which are in cir-

culation all over the world. Now, although there is no doubt that the reading of this kind of literature has often resulted in an attack of hypochondriasis, yet persons free from any morbid predisposition may read these books with impunity. An effect very much like that produced by reading popular medical books may arise from the exclusive occupation of the mind with stories of disease and death, such as are apt to be current in any region where an epidemic disease is prevailing. Such impressions do no harm to the majority of people, but there are a few who become hypochondriacs. It will be readily understood, moreover, that the companionship of a hypochondriac is dangerous to an individual of a hypochondriacal predisposition.

Symptoms and Course.—Hypochondriasis usually develops gradually. At first, there is an indefinite sense of illness, which troubles and oppresses the patient, but which does not as yet disturb his judgment or possess him completely. Nor are this restlessness and distress usually permanent in the beginning. They generally cease at times, and then reappear with somewhat increased intensity. The more profoundly the disease takes root, so much the more assiduously does the patient endeavor to discover the cause of his indisposition. He scrutinizes his tongue, his stools, his urine; he counts his pulse and handles his abdomen. Every trifling irregularity which he perceives, the slightest irritation, the faintest coating of his tongue, a transient colic, an insignificant cough, are all of the utmost importance in his eyes; not because he suffers more than any one else from such symptoms, but because they seem to him to afford a clew to the nature of the grave and obscure imaginary disease. To-day he may dread an apoplexy, to-morrow he may think that he has an ulcer of the stomach; at other times he may imagine his heart diseased, or that he is consumptive, or afflicted with some other serious malady of a character corresponding to his sensations of distress. He studies all the "medical advisers" and other books of "domestic medicine," but, instead of deriving comfort and aid from them, merely finds out new diseases, by which he immediately imagines himself afflicted. As the disease gains mastery over the patient, his belief as to his condition becomes more and more biassed and incorrect. Argument is useless, as it cannot relieve him of his feelings. A few hours only may have elapsed since we last saw the patient. We then may have spared neither time nor trouble in explaining to him that his condition was perfectly free from danger, when a messenger arrives from him, begging us to come in haste, that some serious change has taken place, and that his condition has become a most critical one. In other instances, especially when the patient really has some insignificant disorder, the hypochondriac is not so apt to change his opinion as to the nature of his

disease, but sticks to a belief in one, and cannot be dissuaded from it. He is not like other patients, satisfied with simply complaining of pain, oppression, or fever; partly, because he really feels worse than other people, and, in part, because he is convinced that the doctor "makes light of his sufferings." Hence he exaggerates, and often exhibits the utmost fluency in description of his infinite suffering. In spite of the severity of these imaginary symptoms, however, they by no means despair of recovery; hence hypochondriacs seldom attempt suicide, and never weary of seeking fresh medical advice and of trying new treatment. Sometimes their hopes are so much in the ascendant, and are so productive of happiness to them, that, for a while, in spite of their sense of illness, they are cheerful and in even high spirits. Such intervals, however, are usually very brief, and occur most frequently immediately after the engagement of a new medical man or the commencement of a new "cure." The old mood very soon returns. The false realizations of their sensations, and the erroneous ideas of the patients as to the condition of their own bodies, which we sometimes see in hypochondriasis, are a genuine delirium. Like other insane ideas in other forms of psychical disorder, this proceeds from morbid bias of the mind, and is to be regarded as an attempt to clear it (*Griesinger*). Hallucinations—"sensations originating inwardly"—also arise in hypochondriasis, owing to this sense of illness, and to the attempts of the patient to account for it. Thus the idea that the heart is standing still, or that a limb is withered, or that the body is putrefying, although it is not the result of a genuine sensation incorrectly interpreted, yet it is so vivid that the patients cannot distinguish it from an impression actually furnished by the senses; and they really believe they can feel that the heart does not beat, that the skin is dried up, or that they can smell the putrid emanations from their body. In spite of their mental aberration and morbid fancies, most hypochondriacs are able to transact their business, and to take care of their house and family; and this is the reason why hypochondriasis, usually, has not been regarded as a disease of the mind, being looked upon rather as a nervous disorder, a custom with which we have complied. In the worst forms of the disease, the patient loses all interest for matters which do not bear upon the state of his health. He becomes abstracted, forgetful, and negligent of his affairs, gives himself no further concern about his family, and often remains idle in his bed for years. It is often a long time before his nutritive condition begins to suffer. Gradually, however, especially in bad cases, the patient grows thin, and acquires a sickly appearance, and derangement of the secretory and digestive functions arises. We are not at liberty to account for this emaciation and the other nutritive disorders, by supposing that the

perversion of sensation in the patient's various organs has led to perversion of their functions; for the same emaciation and the same disorder of nutrition may likewise be seen in persons who have fallen into a state of permanent mental depression, as a result of objective conditions, and who feel no morbid sensations in the organs which ultimately become diseased. Moreover, the very improper way in which hypochondriacs often live, and their immoderate use of medicines, contribute a great deal toward the development of a cachectic condition.

Hypochondriasis always runs a chronic course. The cases in which peculiar causes, acting upon a constitutionally timid and imaginative person, induce a transitory belief that he is ill, or a fear that he is about to be ill, are not to be regarded as genuine hypochondriasis. Recovery is not uncommon. More frequently the disease persists throughout life, with varying intensity. It rarely terminates fatally, although there have been instances where the patient died of marasmus and exhaustion.

Treatment.—It is useless to dispute with a hypochondriac, and to try to convince him of the error of his ideas. The only way to cure the patient is to rid him of his morbid sensations. For this purpose it is necessary, in the first place, to correct any existing derangement of the system which, as we have said, is often the exciting cause of the disease, and which would cause a feeling of illness in a sane person. The necessary therapeutic measures vary according to the peculiarity of the case. The benefit often derived from the springs of Karlsbad, Marienbad, and Kissingen, in the treatment of this affection, are, no doubt, mainly due to the beneficial effect exerted by these waters upon diseases of the gastric organs, which so frequently prove a source of hypochondriasis. In other instances, preparations of iron will be appropriate, while the springs will do harm. In others, again, sea-bathing and cold foot-baths are to be used. We must be cautious in the employment of the drastic cathartics, although they can seldom be dispensed with altogether, and although they usually afford the patient a temporary relief; and, above all, we must distinctly warn the patient against over-dosing himself, a practice to which hypochondriacs are very prone. This likewise applies to the use of carminatives, for which the patients nearly always earnestly beg. The object of the psychical treatment, as *Romberg* aptly says, should be diversion of the attention from the sensory to the motor and intellectual spheres. This object will not be attained in educated patients by ordering them to take long walks, to saw wood, to practise gymnastics, and to occupy themselves in other mechanical pursuits, because the attention of the patient is not diverted, by such actions, out of the sphere of sensation.

A hypochondriac merchant, while sawing wood, is always busy with the thought that he is sawing wood because he is sick. Such ordinances, however, especially active gymnastic exercise, are of great service, because, like the cold baths, they induce a vigorous modification in the process of transmutation of the tissues, and because they tend to restore the patient to a sense of physical energy. Diverting amusements, as the excitement which they produce soon becomes extinct, are seldom productive of lasting benefit. Travelling with some definite object in view, or the study of agreeable objects, is much more commendable. Of course, no general rules can be laid down for the fulfilment of the task in question, as our directions must always be in accordance with the capacity, education, and means of the patient.

[CHAPTER VIII.

PARALYSIS AGITANS—SHAKING PALSY.

The most apparent symptoms of this disease are involuntary movements of parts of the body, either a simple trembling or a regular shaking ; sooner or later these are accompanied by partial paralysis of the muscles affected. The disease is uncommon ; it affects chiefly aged men; exceptionally it occurs in middle age, and very rarely in youth. In some cases it has followed severe fright or great anxiety ; in others it has seemed due to unfavorable surroundings, exhausting work, continued exposure to moisture and cold, or to irritation of peripheral nerves from injury ; in other cases no external cause has been found.

The trembling usually begins in the fingers, hands, or arms, and may remain limited there, or may spread to the whole upper or lower limbs ; exceptionally it affects the muscles of the face, larynx, or neck. The tremor may be restricted to the limbs of one side of the body, rarely to one arm and the opposite leg. The movements may cease temporarily, especially in the horizontal position ; but when fully developed it is apt to be continuous, though varying in intensity from mental or physical excitement ; during sleep or anæsthesia the movements cease.

The paralysis may not come on for some time, the impaired use of the limbs meantime being due to disturbance from the motion. Late in the disease there may be a tenseness and rigidity of the muscles, with preponderance for the flexors ; after the tenseness has lasted some time it will cause deformity ; the head may be

drawn down, the arm raised from the thorax, the forearm flexed. If the lower limbs are affected, the rigidity may resemble paraplegia with contractions. There may be various anomalies of sensation, such as numbness, prickling, neuralgia, anæsthesia, heat, etc., and cerebral symptoms, such as headache, dizziness, sleeplessness, etc.

The disease may drag along for years, death at last resulting from age, debility, or some intercurrent disease. The autopsies made have proved nothing about the seat of the disease or the anatomical changes causing it; their results have been so varied that it is not even certain whether the original seat of the disease be in the brain or spine.

Senile tremor, or that of very excitable persons, is less marked than that of paralysis agitans. That due to poisoning by alcohol, lead, or mercury is accompanied by other symptoms of poisoning; but it may be mistaken for multiple sclerosis, although this comes earlier in life, and paresis appears as one of its first symptoms, and the tremor usually comes only when the patient tries to execute some movement.

Although some recent cases have seemed to recover, this must be considered as spontaneous rather than as a triumph of medicine; in some of the cases there may have been an error of diagnosis. Among the remedies said to have proved beneficial are hyoscyamus, hypodermic injection of small doses of Fowler's solution, continued use of carbonate of iron, cold douches while in a warm bath, galvanism, etc.]

[CHAPTER IX.

DELIRIUM TREMENS—MANIA A POTU.

The name delirium tremens is used even by the laity, and unfortunately it is so common that the symptoms which characterize it are very generally known. Frequent as is this affection, the opinions as to its immediate cause are not unanimous. Some believe that it is the direct result of the abuse of alcoholics, while others say that it is due to their sudden withdrawal. It is not improbable that it may be caused either way; perhaps the worst cases are those that come on during the use of liquors; those that result from the patient's inability to obtain liquor, from having a broken leg or some acute disease, or from being locked up, have seemed to us to run a much lighter course than the former. Possibly the long-continued use of alcohol may induce such a state in the gastric mucous membrane that it will absorb neither liquor nor nourishment; this

might make the cases due to direct poisoning worse than those arising from sudden withdrawal of stimulants.

This disease may develop more or less suddenly. It may be preceded by a stage of melancholy, but the actual commencement of the trouble is characterized by marked hallucinations; the patient sees numerous insects or small animals around him; has hairs in his mouth, which he constantly removes with his fingers and spits out; he hears music, voices, etc. While subject to these hallucinations, the patient may still control himself enough to answer questions rationally; but while speaking coherently he will look past you and around the room, and it is evidently by a great effort of will that he keeps his attention fixed on the conversation.

As the disease progresses the harmless animals seen may be replaced by frightful forms, robbers, the devil, and other terrifying personages, who pursue and threaten the poor patient till he is a most pitiable object. At this time more than ever his nerves are unstrung, his limbs in a perfect quiver from excitement and fear, perspiration bedews his face, the tongue is dry, and he constantly stares around as if following his persecutors with his eyes. He cannot sleep or eat. A good nurse, or a courageous, vigorous friend, may persuade him to drink milk, beef-tea, etc.; but the power of masticating and of supplying saliva for solid food seems to be absent.

In the first attack very frequently, and in subsequent ones more rarely, the above symptoms will only last a couple of days; then the patient will eat a good beef-steak, have a sound sleep, and subsequently be well except for a slight gastric catarrh, which is very apt to have been induced by his previous prolonged use of liquor. In severe cases, after one or several days of the delirium, while the patient does not seem to be dangerously ill, he may suddenly die from paralysis of the heart; or in trying to escape his imaginary tormentors he may jump from a window or otherwise do himself serious injury. If the disease lasts several days, with delirium and inability to eat, the tongue becomes dry, the pulse frequent and dicrotic, and the patient dies in a typhoid state. In these cases there is usually chronic disease of the kidneys and liver; evidences of chronic inflammation of these organs, and perhaps of the cerebral meninges, being the chief changes found on autopsy.

Delirium tremens rarely lasts a fortnight; in our own experience those cases that last over six days end in recovery. It must be regarded as a very treacherous disease, and our prognosis may always be guarded till the patient has eaten and retained some safe solid food, such as tender beef or mutton.

In all cases of delirium tremens the condition of the kidneys and of the thoracic organs should be carefully scrutinized; for we should bear in mind that pneumonia attacking a person of intemperate habits presents all the symptoms of delirium tremens, and in many such cases there is neither cough nor expectoration, nor does the patient complain of pain, nor indeed of any symptom likely to direct the attention of the physician to the real nature of the disease. It has not unfrequently happened that a man has died under diagnosis of delirium tremens, and a pneumonia, which had not been even thought of during the progress of the case, has been revealed upon dissection.

The symptoms of Bright's disease also are not always so marked as to attract immediate attention. A uræmic convulsion sometimes admonishes the unwary practitioner that a grave condition of the kidneys has been overlooked.

Treatment of delirium tremens, as of any disease, must depend greatly on the symptoms; but numerous special plans have had their advocates, such as that by opium, alcohol, and digitalis; each of these has doubtless proved useful in the hands of its advocates, but no one has proved universally acceptable. The remedy at present most generally relied on is bromide of potassium. It may be given in doses of thirty grains every hour till the patient sleeps; if a few doses do not accomplish the desired result, an equal quantity of hydrate of chloral may be added to it. When a good sleep has been secured, the hallucinations may not recur, and further treatment may be unnecessary, except such as is required for the general state of the patient.

Another remedy which is very quieting, and may be used subcutaneously when there is difficulty in getting the patient to take bromide of potassium, is hyoscyamin, which may be given in doses of the twenty-fourth of a grain, and if necessary increased to the twelfth or sixth of a grain.

When the patient comes under treatment with a stomach full of alcohol and undigested food, "just off a spree," it is well to give a mild emetic and a cathartic (perhaps calomel); then, unless there are complications, to rely greatly on a good-natured, stout, resolute nurse, and good diet. If the symptoms be not urgent, we may give the mineral acids, Horsford's acid phosphates, and similar remedies. If there is marked sleeplessness, we may try one of the above remedies. An important question is whether the patient shall have any alcoholic stimulant. If the stomach is quiet, and apparently in a condition to digest food which the patient objects to taking, it may

be well to promise a drink if he will eat a piece of steak; but as a matter of treatment, or by way of "tapering off," we may generally reject alcoholics.

When a quiet sleep has been secured, and a meal of solid food has been eaten and retained, the patient generally progresses favorably.]

[CHAPTER X.

MORPHINE HABIT—OPIUM-EATING.

Closely allied to chronic poisoning by alcohol, with its frequent accompaniment delirium tremens, is the result of long-continued use of opium or the salts of morphine. This habit is not uncommon, and as it is generally begun under the physician's recommendation, we may consider ourselves as responsible for its cure.

As a patient suffering from indigestion finds his pain relieved by alcoholic stimulants, he resorts to them more and more frequently, and finally employs them to excess; in the same way frequent attacks of pain from neuralgia, rheumatism, or inflammation induce repetition of the doses of narcotics that have once been found efficacious. Hence it would seem imperative that, when prescribing the various pain-quellers, we should warn the patient against too frequently resorting to them, and explain to him that they all gradually lose their effect, and after being used for a time cease to afford relief, or must be given in gradually increasing doses. The preparations of opium being especially liable to abuse in this way, we may picture to the patient the painful symptoms of chronic poisoning that are likely to result from their continued use, and which may finally prove more annoying than the original pains for which the anodyne was given. It is also claimed that protracted use of opium begets a special tendency to neuralgia, and demoralizes the entire nervous system.

There is no doubt that many opium-eaters reach advanced age under the influence of that drug; many of those who drink intoxicating liquors, even to excess, live to be quite old, and many aged men have been smokers from their youth; but, as a rule, free and continued use of opium, alcohol, or tobacco will be followed by their peculiar toxic effects. It seems that any of these articles may be used with greater impunity on a full stomach; and those who habitually indulge in them only after a full meal are less liable to suffer bad results.

The symptoms of opium-eating or morphine habit are not always very marked, even where the patient's life is rendered a burden by it; they vary greatly in relation to the time which has elapsed since the dose last taken. While the effect is recent, and the nerves are "keyed up," the patient may be lively and unusually bright; as the effect begins to wear off, there comes a quiet, rather drowsy time, and those who are entirely regardless of the results do not go beyond this stage while their digestion and nerves will stand repetition of the dose, for they at once resort to their stimulus. But if limited means, a troublesome conscience, or other cause prevents the use of more than a certain quantity daily, the second stage is followed by a third, where we have what are usually given as the symptoms of opium habit, viz.: sunken features (as if the skin were drawn tightly over the cheek-bones), lack of healthy color, furred tongue, constipation, loss of appetite, sleeplessness, shaky hands covered with clammy moisture, and great mental depression or restless longing for more of the drug. Cold hands and feet, tendency to sweat, and complaints of most varied pains and discomforts are common. An almost universal symptom is emaciation, which is magnified by the sunken look above referred to. Mentally the patients are depressed, suffer from loss of memory and energy, and are apt to be untrustworthy.

If the use of the drug be suddenly and completely stopped, a state resembling delirium tremens may supervene and result in death. In strong patients, however, this sudden arrest is one of the modes of treatment recommended; but if we have the earnest cooperation of the patient, or have complete control of him, we may pretty certainly break off the habit without this rude shock, by gradually reducing the amount of the drug, substituting for it if necessary a *variety* of other remedies, not relying on any one for a length of time for fear of replacing the opium habit by some other only a little less pernicious. During the period of withdrawal the doses of opium given will have to be regulated to some extent by the previous habits of the patient; i. e., if he has taken his dose only at night, we have simply to reduce this dose gradually (without the patient's knowledge). If it has been taken more frequently, it is well to give a dose of the opiate with a tonic shortly before the midday meal; this will enable the patient to eat well, and possibly the effects may last till the evening meal. If we can get along with two doses daily, the second dose should be given about bedtime. Administering the two doses in this way gives the best chance for plenty of nourishment and plenty of sleep, which will be found

absolutely necessary to keep the patient's "courage to the sticking-point" of resisting the craving which exists even when the dose is not being reduced. During the treatment appetizing food should be offered and mental and physical occupation provided. Addition of atropin to the opiate, occasional hot baths for the extremities, and frictions with alcohol, seem to check the pain and sweating in the hands and feet.]

ADDITIONS TO THE REVISED EDITION OF 1880.

SECTION IV. — GENERAL NEUROSES, OF UNKNOWN ANATOMICAL ORIGIN.

1.—P. 405.

Occasionally the urine passed a few hours after an epileptic seizure contains albumen. Sometimes there is temporary mental perturbation instead of a regular epileptic fit. The seizures may be followed by paresis or paralysis, in which case we may suspect disease of the brain ; or there may be aphasia, aphonia, dysphagia, asthenia, etc.

The investigations of *Reynolds* oppose the general view that the disturbance of intelligence is in direct proportion to the number and severity of the seizures. *Nothnagel* even thinks that the mental disturbance in the majority of cases is not the result of changes in the pons and medulla oblongata, but accompanies them ; i. e., the epilepsy and psycopathia are both due to some neuropathic disposition.

It is always important to decide whether any given case is idiopathic or secondary to some other disease, and this must be determined by a careful examination of the whole body and of the surrounding circumstances. We may suspect organic disease of the brain when there is a permanent partial spasm, or when it always begins at the same part of the body.

Attention has been called to pain in the hypogastric region, which was attributed to the ovary ; this pain may be spontaneous or induced by pressure over one or both ovaries (oftenest the left). It is said that an hysterical aura is often induced by this pressure —a feeling of constriction about the stomach and neck, nausea, palpitation, tinnitus, disturbance of vision on the side of the pain-

ful ovary; and that further pressure upon the affected part may induce a true hysterical attack.

The following is said to be a favorite prescription with Dr. *Brown-Séquard:* Bromides of sodium, iodinium, and ammonium, each ʒ iij; iodides of potassium and ammonium, each ʒ iss.; ammon. sesquicarb. ʒ j; tinct. calumbæ ℥ iss; aquæ dest. ad ℥ viij. S. teaspoonful and a half before each meal and three teaspoonfuls at bedtime. If bromide of potassium fails, try conium, cannabis indica, or oxide of zinc.

Chapman's ice-bags applied to the spine for two to eighteen hours have proved useful in some cases. Electricity, even in the constant current, has not fulfilled expectations; when used as an adjunct, we try to affect the pons and medulla oblongata by placing the electrodes on each side of the neck; or we may galvanize the sympathetic or peripheral nerves from which there is any reflex excitement.

Dr. *S. W. Mitchell* recommends inhalations of from two to five drops of nitrite of amyl for averting attacks; but they can only be tried where there is a protracted aura, and in cases where the face becomes pale at the onset, showing spasm of the vessels. Sometimes this remedy seems not only to avert attacks, but to render them less frequent and severe; in other cases the escape was followed by severe attacks.

2.—P. 418.

Hysterical spasms may resemble epilepsy, or the latter may occur in hysterical patients. There may be hysterical paralysis of almost any muscles, but especially of those of the extremities. The electro-muscular contractility remains normal; in most cases there is cutaneous anæsthesia. Rarely with paralysis of a limb there is facial paralysis, anæsthesia of the skin of the face, ptosis, etc. There may be paralysis of the pharynx or œsophagus, which may be diagnosed from spastic dysphagia by the passage of a sound. Sometimes enormous tympanites occurs, the accumulation of gas seeming due to temporary arrest of the gastric and intestinal contractions; the same sluggishness of peristaltic action may cause constipation. Aphonia, due to paralysis of the glottis muscles, is seen quite often. In rare instances paralysis of the diaphragm has been seen, when on respiration the movements were in the reverse direction, viz., sinking in inspiration and protruding in expiration. There may be paralysis of the bladder with hemi- or paraple-

gia. These paralyses may last for days or weeks, and then suddenly disappear.

There may be great variation in the heart's action—palpitation with a feeling of fulness and pressure in the chest, of constriction and anxiety ; or, on the contrary, a tendency to temporary lack of action, and faintness with hysterical trance.

There are changes in the vessels, the skin changing from red to pale, either on part of or the entire body. These changes of the vessels may affect different organs, as brain, lungs, uterus, etc., or cause bleeding from the nose, mouth, stomach, respiratory passages, etc. But hysterical patients sometimes simulate such things, drinking blood and vomiting it, causing bleeding from the skin, etc.

DISEASES OF THE SKIN.

No notice has been taken, in the following chapters, of the alterations which the skin undergoes in the acute and chronic infectious diseases. Like other symptoms of measles, scarlatina, small-pox, typhus, and syphilis, these cutaneous affections are to receive the attention due them, when we come to discuss the subject of the infectious diseases themselves, as they only form a single link in the chain of nutritive disturbances to which these maladies give rise.

We classify diseases of the skin as we do diseases of other organs, according to the anatomical nature of the lesion which the disease produces. We shall, therefore, have to speak of hypertrophy, atrophy, hyperæmia, anæmia, hæmorrhages, inflammations, neoplastic growths and parasites of the skin. Since, however, it is in our power to observe the variety in extent, in intensity of the morbid process, more accurately upon the skin than upon other organs, and as we are able directly to watch certain anomalies of secretions which are not accompanied by palpable change of structure, diseases of the skin admit of a much more minute classification than is possible in diseases of other organs. We must so far conform to the practice of applying names to the cutaneous affections other than those belonging to the analogous lesions upon other parts of the body, as to associate their proper pathological and anatomical title with their customary name. Of the unpractical and useless classification of the various forms of skin-disease into innumerable subdivisions, we shall merely make cursory mention.

I.—*HYPERTROPHY OF THE SKIN.*

An hypertrophy, involving all the component parts of the skin, the connective tissue, the vessels, nerves, epidermis, hair, and glands, only occurs in small isolated spots as a congenital anomaly. Most of the prominent "mother's marks" belong to this class, as well as the

soft cutaneous warts or moles. But, even here, the hypertrophy does not involve all the tissues of the skin in equal degree. In the majority of the projecting "mother's marks" and moles, there is a predominant development of pigment and hair; their brown or blackish color and the strong growth of hair with which they are covered being one of their striking features.

Large accumulations of horny epidermic cells are often found upon certain spots of the body; the callosities, the corns (*clavi*), and the cutaneous horns, originate in this way. *Callosities* consist of low flat, horny elevations, of a rounded or irregular form; the condition of the cutis which they cover being either normal or slightly hyperæmic. Callosities are most apt to form at points exposed to irregular pressure. Hence they are found upon the heels and upon the soles of the feet of most persons; upon the hands of blacksmiths, locksmiths, and other mechanics; and upon the index-fingers of tailors and of seamstresses. *Corns* are small, but very hard and thick conical callosities, induced by the pressure of the boots or shoes, and which cause a circumscribed atrophy of the skin. The so-called cutaneous horns consist in an excessive hypertrophy of the epidermis over a circumscribed spot of skin. Some horns do not spring from the papillæ of the skin, but develop from dilated hair-follicles, and may be regarded as monstrous hairs. There is also a diffuse hypertrophy of the epidermis, which is accompanied by hypertrophic development of the papillæ of the skin. Of this affection, which, when of moderate intensity, is called *pityriasis*, and when more severe is called *ichthyosis*, we shall treat more in detail hereafter.

The brunette complexion, congenital in some persons, is due to a copious deposit of pigment in the cells of the rete Malpighii. Another congenital anomaly, which is seen in many persons, consists in a heavy deposit of pigment in circumscribed patches upon the rete Malpighii, producing brown or black spots (*chloasmata*, *melasmata*). When the patches are of wider circumference, they are called pigmentary nævi (*nævus spilus*). If only as large as a lentil, they are called ephelides or lentigines (Leber-flecke). The pigmentary marks and spots, which are unaccompanied by hypertrophy of the cutis, and which, therefore, do not project above the surface of the skin, are likewise often covered with hair. In the majority of persons the formation of pigment in the rete Malpighii is increased under the influence of the sun's light and heat, and under that of wind and dampness; hence, among soldiers, field-laborers, and particularly among seafaring persons, the exposed portions of the skin assume a uniform brown color. It is remarkable, too, that in some persons irritation of this kind does not produce such deposit of pigment, or, as people say, they do not become "sunburnt"

or "tanned." There is another circumstance, too, which is very difficult of explanation, namely, that in certain persons with very white skins, especially in blondes and in red-haired individuals, the pigment formed under the influences just mentioned is only deposited in small circumscribed specks and spots upon the unprotected surface. In summer, the hands, face, and arms of such persons, though screened from the direct rays of the sun, become covered with rounded spots of a more or less dark color, called "freckles" (*ephelides*). The bronzed skin of a seaman gradually loses its deep color if he stays at home in winter, and, under similar circumstances, freckles also fade or entirely disappear. Freckles may be removed by means of applications capable of producing desquamation of the epidermis, together with its deeper pigmentary layer; but, if the exciting cause be continued, they will return in course of a few weeks. The well-known Lilionèse is merely a palliative cosmetic, as is also the wash recommended by *Hebra* (hydrarg. chlor. corrosiv. gr. v; aquæ ℥ j). The application is only to be kept on for a few hours, and care must be taken that the compress upon which the solution is applied is free from folds. If the skin becomes much inflamed, it is to be covered by compresses dipped in oil. In a few days, as the epidermis scales off, the freckles disappear.

In pregnant women, and women suffering from disease of the sexual organs, brown spots often appear upon the face, especially upon the forehead and upper lip (*chloasmata uterina*). In most women they disappear some time after confinement, but in some they are very persistent or even become permanent. This phenomenon is as inexplicable as is the increase in the pigmentation of the rete Malpighii, and of the areola of the nipples and skin of the linea alba during pregnancy.

Besides the diffuse hypertrophy of the papillary portion of the skin, which occurs in ichthyosis, hypertrophy of a single papilla, with hyperplasia of the epidermis which covers it, is of very common occurrence. It is in this manner that warts and condylomata are formed. Warts arise by the elongation and combination of a few papillæ, so as to form a cone. It is covered by a very thick and hard layer of epidermis. If the several papillæ of which the wart consists be each covered separately by its epidermic envelope, the wart has a cloven fibrous appearance. The cause of these growths is unknown. Uncleanliness of habit certainly plays but a very subordinate rôle in their production, for, even among the cleanest people, the skin, especially that of the hands, often becomes covered by them in a short time. The sudden manner in which this papillary hypertrophy often disappears is equally puzzling. The laity are accustomed to ascribe the

disappearance of warts to the action of the so-called sympathetic remedies or charms. Condylomata differ from common warts, as their papillæ not only grow longer, but they also throw out lateral offshoots, and the epidermis which covers them is not so thick and tough. There are two forms of them, the pointed and the broad. The former appear upon the mucous membrane of the external genitals, the urethra and vagina, as well as upon parts of the skin which have been moistened by the vaginal or urethral blenorrhœal secretion. Their appearance is that of a mulberry or cauliflower, or, if compressed laterally, they look like a cock's comb. Pointed condylomata require local treatment. The structure of the wide condylomata is very like that of the pointed form. They are less prominent, however, and show a tendency to superficial ulceration. The most common seat of the broad condylomata is upon the labia, the scrotum, and between the nates. More rarely they occur upon the lips and between the toes. As they are the consequence of constitutional disease, they require general antisyphilitic treatment for their cure, instead of local applications.

The so-called *polypi* of the skin, and the hard and sometimes pedunculated tumor, known as *molluscum simplex* (*fibroma molluscum* of *Virchow*), are results of a circumscribed hypertrophy of the connective tissue of the skin. The so-called *keloid* is a peculiar form of partial hypertrophy of the cutis, resulting in the formation of irregular tumors, which in structure resemble a scar. *Pachydermy*, or *elephantiasis Arabum*, is due to a diffuse hypertrophy of the cutis and subcutaneous connective tissue. Of this we shall speak more fully hereafter.

The hypertrophic development of the capillaries of the cutis, which sometimes is combined with an hypertrophy of its connective tissue, gives rise to the formation of red or reddish-brown spots, or tumors in the skin, called *telangiectasis*. They may be congenital (*nævi vasculares*), or they may not form until some time after birth. Telangiectasis must be regarded as of two kinds—a kind which, after attaining a certain size, remains stationary; and a kind which grows continually, its capillaries finally becoming so much dilated as to give way and to bleed profusely.

The majority of "mothers' marks" are nearly always complicated with hypertrophic development of the hairs and cutaneous glands upon small portions of the skin. Curious cases are also met with in which there is an extraordinary premature development of the beard, and of the hair upon the pubis, and sometimes a remarkable growth of hair covering the whole body, or parts of it, no other irregularity coexisting. *Molluscum contagiosum* (*epithelioma molluscum* of *Virchow*) is the result of hypertrophy and dilatation of the hair-follicles,

which become distended by a collection of epithelial scales and globules of a fatty lustre. The tumors, which at first are scarcely larger than a pea, and are covered by the normal skin, gradually enlarge, while the skin becomes tense, red, and depressed in the middle, like a funnel. New nodules develop in the vicinity of the first one, so that finally a large area of the skin is often covered by these molluscous tumors. This continuous progress, and the communication of the disease to other persons, the actual occurrence of which has been well authenticated in several instances, indicate that it is of a contagious character. The bearer of the contagion seems to be the fatty globules above mentioned.

CHAPTER I.

DIFFUSE HYPERTROPHY OF THE PAPILLARY LAYER AND OF THE EPIDERMIS—ICHTHYOSIS.

Etiology.—It has been stated already that the exuberant formation of epidermis, which takes place in ichthyosis, is due to an abnormal development of the papillary layer of the matrix of the epidermis. *Bärensprung*, of whose excellent work upon cutaneous diseases we have made free use whenever the publications of this profound investigator have been available, makes a distinction between congenital ichthyosis in its narrowest sense, where the child comes into the world clad in a thick horny corselet, and the true ichthyosis. In the former affection the child is born dead, or dies soon after birth, and it would appear that the horny case which covers it must have formed at an early period of intra-uterine life, probably through melting together of the caseous varnish composed of cells of epidermis and cutaneous secretion. It is always evident that the rigid horny coat, which is all cracked into fragments, has become too small to cover the fully-grown fœtus, and has crippled the development of its nose, lips, ears, fingers, and toes.

The papillary hypertrophy, which is the cause of the true ichthyosis, likewise appears to be an hereditary and congenital disease. The fact that the malady nearly always remains undetected during the first year of the child's life, is to be ascribed to the manner in which the skin is cared for during that time. The family history of a patient with ichthyosis often shows that other members of the family, brothers, parents, or grandparents, or other relatives, have suffered from the same disease.

Besides the congenital true ichthyosis, which usually extends over the entire surface of the body, there is a milder and acquired form

of the disease, which remains confined to portions only of the skin, and which complicates pachydermy.

Symptoms and Course.—Instead of presenting the smooth appearance usually seen upon the skin of a healthy person, the skin in the milder grades of ichthyosis is roughened and covered with delicate white scales. It is this milder form of the disease which is usually called pityriasis. The term pityriasis, in this sense, signifies a desquamation of the cuticle depending upon a mere exuberant growth of epidermis, and not upon other disease. We know such to be the case, from the habitual presence of the conditions above described, and by the absence of any symptoms of congestion or of inflammation, or of any derangement in the secretion of the glands and sebaceous follicles. Most cases of desquamation of the epidermis upon the head, in which the fine white scales adhere to the hair, and cover the collar of the coat, do not depend upon exuberant formation of epidermis, but upon a superficial dermatitis. The scaling off of the skin of the palms of the hands and soles of the feet, too, is generally due to superficial inflammation of the skin, and will be discussed more in detail in the chapter upon eczema. In genuine ichthyosis, the epidermis is detached in the form of larger and thicker scales, usually stained of a dark color by pigment and dirt. In the worst forms of the disease the epidermis is in horny plates, and even forms warty or spinous projections. Hence a great number of subordinate varieties of ichthyosis have been recognized, ichthyosis simplex, cornea, hystrix, etc., which, however, are not varieties in kind, but merely in the degree of the disease. Certain parts of the body, the face, the palms of the hands, the soles of the feet, the armpits, and the bends of the knees and elbows, are not attacked by the affection, while its favorite seat is upon the dorsal surface of the extremities, especially upon that of the knees and elbows. *Hebra* accounts for the fact that we cannot, at first, detect the existence of ichthyosis in newly-born children, upon the ground that a child in the womb of its mother is in a continual warm bath, which keeps the epidermis in a state of maceration. Moreover, owing to the continual necessity for washing little children in their first year, the scales of epidermis are not allowed to collect, and hence the disease usually remains undetected.

Treatment.—This disease is incurable, as we know of no remedy capable of producing involution of the hypertrophy of the papillary layer of the skin. Our experience as to the uselessness of arsenic, antimony, tar, and other external and internal medicines, is too ample to warrant our making further trial of them. It is well to advise the patient to take a warm bath daily, with or without the addition of an alkali, and diligently to anoint his skin with some unctuous substance,

as by these means the extensive accumulation of detached epiderm s may be prevented.

CHAPTER II.

DIFFUSE HYPERTROPHY OF THE SKIN AND SUBCUTANEOUS CONNECTIVE TISSUE—PACHYDERMIA—ELEPHANTIASIS ARABUM.

Etiology.—Repeated inflammation of the skin, and especially repeated and permanent obstruction of the veins or lymphatics of a part, sometimes lead to enormous growth of the connective tissue of the skin, subcutaneous and intermuscular substance, and even of the periosteum of the bones of the affected part. This condition is called *pachydermia*, or, from the clumsy, deformed appearance of the limb attacked, it is also called *elephantiasis arabum*. This disease has nothing in common with the *elephantiasis græcorum*, or leprosy (*lepra*, *Spedalskhed*). We do not know the reason why it is that a case of repeated dermatitis, or phlebitis and primary thrombosis, or of obliteration of a vein or lymphatic, only now and then terminates in pachydermia, and why it does not always occur. It is also quite strange that this disease should be much more common in certain countries, especially in the torrid zone (Barbadoes leg), than it is with us.

Anatomical Appearances.—The most usual seat of the disease is upon the leg, which undergoes more or less irregular enlargement, and attains more than two or three times its normal size. The skin is immovable, and, when the papillary layer also takes part in the hypertrophy, it is covered with scaly crusts. The tissue of the skin, and of the adipose layer below it, is converted into a dense firm mass, resembling bacon, whose microscopic elements consist of connective-tissue cells of different stages of development. The muscles are atrophied and in a state of fatty degeneration, owing to a want of exercise, and to the pressure to which they are subjected by their surrounding connective tissue, which is likewise involved in the thickening. The veins and lymphatics are often obliterated, and there is frequently a varicose enlargement of them below the point of obstruction. The anatomical appearances are quite similar when the disease attacks the upper extremity, the scrotum, the penis, or the labia majora.

Symptoms and Course.—The first symptom of the disease is either an erysipelatous inflammation of the skin, or else a lymphangitis or a phlebitis. Some authorities state that the local disease is often preceded by violent fever. This cannot be regarded as at all singular, as in other inflammations the febrile disturbance is generally most severe at the commencement of the attack, and attracts attention be

fore the functional disease becomes apparent. The portion of the body which swells during the inflammation does not return to its normal size after the fever subsides, but remains moderately enlarged, and assumes a soft, boggy consistence. Soon, usually within a few months, another attack occurs, which runs the same course as the first one, and results in a further enlargement of the affected part. As the attacks are repeated, and as the intervals between them diminish, the deformity and weight of the limb increase, and the doughiness, which is a result of œdematous infiltration, gives place to a board-like hardness, due to the formation of dense connective tissue. If, as the inflammation recurs, the pachydermy spread beyond the point originally attacked, all the different grades of the disease are often exhibited simultaneously in the same extremity. Excepting during the periods of inflammation, there is no pain in the swollen part. It is often the seat of a superficial (eczematous) inflammation of the skin, accompanied by a liquid exudation beneath the epidermis and upon the free surface. The mobility of the afflicted member is, of course, seriously impaired.

Treatment.—A suitable treatment will often afford great relief in cases of pachydermia which have not advanced too far, and may even effect a complete cure. During the initiatory stage of inflammation the limb must be elevated, and this posture must be retained for some time after the inflammation has subsided. Cold applications are then to be made. The inunction of mercurial ointment is also urgently recommended in this stage. After the inflammation has subsided, we must proceed to methodical compression of the part. *Hebra* recommends that the limb be enveloped in a cotton bandage previously soaked in water. This is to be applied from the toes upward, so that each turn of the bandage shall almost cover the preceding one. It may be drawn very tightly, as the patient can bear a very firm pressure without inconvenience, and as even a very tight bandage usually becomes loose in a few hours. This simple treatment, if kept up methodically, often produces a most beneficial effect.

In elephantiasis scroti, in which the scrotum sometimes reaches down as low as the knees, and weighs over a hundred weight, and in elephantiasis of the labia majora, the tumor must be removed by the knife.

II.—*ATROPHY OF THE SKIN.*

Atrophy of the skin may appear as one of the symptoms of a general marasmus, and not only in the senile marasmus, but also in that premature general atrophy induced by exhausting illness. Upon pinching up the skin of a marantic individual into a fold, or, upon cut-

ting it in *post-mortem* examination, a striking diminution of its thickness will be observed. A coating of detached scales of epidermis is usually found upon the skin of such subjects. This is not a product of any coexisting hypertrophy of the epidermic layer, but has formed simply because the cutaneous secretion is diminished, which keeps the skin pliable during health, and which causes the epidermis of healthy persons to fall imperceptibly. In other words, in the so-called *pityriasis tabescentium* the epidermic cells are not generated in undue quantity, but they are shed in a more conspicuous manner than is natural to a person in good health. Atrophy of the skin may also proceed from continued pressure either from within or from without. We have already said that a corn induces partial atrophy of the corium. Favus crusts and scabs, which have adhered to the skin for a long time, have a similar effect. As pressure from without sometimes injures the papillary layer of the skin, the product of the latter, the epidermic cells, is formed in diminished quantity, and hence the cuticle over the compressed point is remarkably thin. The effect is different when the pressure upon the skin proceeds from within, as, for instance, in the great distention of the belly which occurs in pregnancy or during dropsical effusion, and in cases of excessive swelling of other parts of the body covered by skin. In such cases it is the deeper layers of the corium and the glandular structure of the skin which suffer, the production of epidermis not undergoing any derangement. I have no doubt that the pityriasis of the abdomen and extremities consequent upon repeated pregnancy and extensive dropsy, as well as pityriasis tabescentium, is attributable to atrophy of the deeper layers of the corium and cutaneous glands, with abnormal dryness of the epidermis.

A congenital absence of pigment throughout the rete Malpighii of the whole body occurs in albinos. Sometimes from unknown causes the pigment disappears from circumscribed patches of skin. These spots are of a milky whiteness, and, as they most frequently occur in individuals of a strongly-marked brunette complexion, they create a striking contrast with the surrounding surface (vitiligo, achroma).

The hair-follicles often undergo atrophy, particularly the follicles of the scalp, and, in consequence of this, the hair falls off. If the atrophy does not cause complete destruction of the hair-bulb, the production of hair does not cease entirely, but the wasted follicles can only produce a fine woolly down, instead of vigorous normal hair. Bald-headed persons, finding this downy growth upon their heads, are often inspired with fallacious hopes, especially if just before they have been using the "eau de lob" or "lion-pomade," and have faith in the tales told of these articles. Baldness arising from atrophy of the hair-bulbs is called *calvities*, or *calvities senilis*, from its liability to occur in old per

sons. Not unfrequently, however, this calvities occurs in younger persons; hereditary predisposition apparently being one of the most common of its causes. No reliance is to be placed upon the statement of baldness's having arisen from over-exertion of the brain, over-anxiety, or from sexual excess. There is a very large number of profoundly-learned persons, as well as careworn and dissipated people, whose hair grows luxuriantly; while many others, who think but little, and live continently and unoppressed by care, lose their hair early in life. Of course, there is no means of producing a development of new hair-bulbs; hence, in spite of the pretensions of charlatans, this form of baldness is incurable. This is not the case with the *defluvium capillorum*, or falling out of the hair, which occurs during and after certain acute and chronic diseases, during which the hair-follicles suffer temporary derangement of nutrition. Here the follicle is not destroyed, nor is it even permanently injured. When the disease causing the loss of the hair has subsided, and when its effects upon the general constitution have disappeared, the hair-follicles are restored to health, and reproduce new hairs in place of the fallen ones. Among the acute diseases, typhus, and of the chronic ones, syphilis, are the most frequent causes of defluvium capillorum, as this latter form of falling of the hair is called. Severe pneumonia, however, and nearly every other exhausting disease, and in a slighter degree the puerperal state, almost always occasion more or less loss of the hair. *Alopecia circumscripta seu area Celsi* also seems to depend upon temporary derangement of the nutrition of the hair-follicles. In this complaint, which is not uncommon, round spots of variable size appear upon the head, or, as is less usual, upon the beard, or upon other regions, in which the hairs break off close above the roots, split into brushes, and fall out, so as eventually to form a bald spot closely surrounded by a dense growth of hair. The skin of the bald spot appears perfectly healthy, and this will readily enable us to distinguish alopecia circumscripta from *herpes tonsurans*, which may also produce round bald spots upon the skin. The cause of alopecia circumscripta is unknown. It is not due to the presence of a vegetable parasite. After a while the bald spot is once more covered with healthy hair. Loss of the hair induced by inflammation of the scalp, and by parasites (favus, herpes tondens), is to be treated of by-and-by in its appropriate chapter.

In very old persons the hair almost always loses its color, and, according to the observations of *Seitz*, the discoloration begins at the tip, and extends rapidly—i. e., in a few days—over the whole hair. Now and then the hair loses color throughout its whole extent from the first. The hair of young persons also loses its color sometimes, and it then often takes place more rapidly than is usually

the case among older persons. As in premature baldness, hereditary tendency seems to be the main cause of premature grayness of the hair, although, perhaps, care and sorrow may also play some part in producing the change. It is a curious fact that a hair, which is only gray at its tip, if pulled out, does not become gray throughout, but remains permanently in the same condition. From this circumstance it would seem that the matrix exerts some vital influence in rendering the hair gray. This fact, moreover, renders those stories, which have been handed down to us, far less incredible, although their truth has been much doubted, of the hairs becoming gray in a few days from the effect of intense mental emotion; all the more so, since, according to *Pfaff*, the grayness does not depend upon a disappearance of the pigment from the hair, but upon a thickening of its cortical layer. In a case reported by *Landois*, sudden grayness of the hair took place in consequence of an increased development of air in the hair. The pigment does not disappear from the medullary substance of the hair until in advanced old age.

III.—*HYPERÆMIA AND ANÆMIA OF THE SKIN.*

The quantity of blood contained by the skin varies more than that contained by any other organ, because the skin is far more exposed than other parts of the body to influences capable of modifying the circulation. In our first volume we have already given a detailed account of those abnormities of the cutaneous circulation which arise from disease of the heart, including both the overloading of the arteries and arterial capillaries from increased cardiac action, and the engorgement of the veins and venous capillaries (cyanosis), which results from lack of power in the heart.

Active hyperæmia of the skin (fluxion) occurs upon exposure of the skin to great heat, especially to heat and moisture combined. It also may result from the application upon it of irritating substances, such as cantharides, mustard, or spurge-laurel, or from mechanical injury. In all such cases, as has repeatedly been mentioned, the first effect of the irritant seems to be a relaxation of the cutaneous tissue; and a dilatation of the capillaries seems to be consequent upon the reduced power of resistance of the tissues which surround them. If the hyperæmia be intense enough to produce a redness of the skin, visible through the epidermis which covers it, it is called *erythema*, and is further distinguished as erythema *caloricum*, *solare*, *venenale*, according as the redness proceeds from heat, the sun's rays, or the action of mustard, cantharides, or spurge, and the like. With equal reason, redness

of the surface, caused by a blow, might be called erythema traumaticum. Such a variety of names for hyperæmia of the skin is quite useless, and, as some of the inflammatory cutaneous affections, when accompanied by redness, are also called erythema, it only creates misunderstanding and confusion. Partial hyperæmia of the skin, moreover, is the first symptom of most of the acute and chronic exanthemata, in whose subsequent course exudation is thrown out either within the skin or upon its surface. Finally, partial cutaneous hyperæmia is very often observed in certain febrile diseases, without our being able to account for its occurrence. In such cases the hyperæmia usually is limited to a very small circumscribed area, forming rounded or irregular red spots, varying in size from that of a lentil to that of a farthing, and is thus called roseola. This name, however, is not applied exclusively to the red spots of simple hyperæmia (*macula*), but also to the small red nodules arising from an infiltration of the skin combined with hyperæmia (*papula*). Typhoid fever, the typhoid stage of cholera, and other infectious disorders, are accompanied by roseola, but it also is by no means rare in the febrile, gastric, and intestinal catarrhs of children, as well as in inflammatory diseases of the brain and lungs. When we can discover a cause for the fever, we call the roseola a *symptomatic roseola;* when no cause for the fever can be found, the eruption is said to be *idiopathic*. To the latter class belong the so-called roseola æstiva, ros. autumnalis, ros. infantilis, and some of the affections entitled measles ("Rötheln").

The only symptoms of erythema are reddening of the skin and an augmented sense of heat. The hyperæmic surface becomes blanched if the blood be expelled from the skin by pressure, while the redness arising from hæmorrhage into the cutis is not effaced by pressure. When the reddened spot is likewise much swollen and painful; when the pressure leaves a white spot instead of a yellow one behind; when, after the redness subsides, the epidermis scales off, the disease is not simple hyperæmia, but inflammation with infiltration.

Anæmia of the skin is an important symptom of poverty of the blood. It also arises from exposure of the skin to a very low temperature, both owing to the physical action of cold and to the contraction of the cutaneous muscles to which cold gives rise. Both of these effects render the skin denser and more resisting, and thereby impede the influx of blood into the capillaries. This effect is increased by the contraction of the smaller arteries which takes place at the same time. Finally, a partial anæmia or *ischæmia* of the skin may arise independently of the influence of cold, from spasmodic (?) contraction of the cutaneous muscles and muscles of the arterial walls. This phenomenon is most frequently observed during the rigor which ushers in a

fever. Sometimes, however, it occurs spontaneously, and without assignable cause, as is seen in the so-called "deadness" of the fingers and toes.

Hyperæmia and anæmia of the skin seldom require active treatment. The application of cold is indicated in the former, and, in the latter, warmth, combined with moisture, as well as the application of cutaneous stimulants and stimulating friction, is the proper remedy.

IV.—*INFLAMMATION OF THE SKIN.*

Under this heading we treat of all the nutritive disturbances of the skin in which an exudation is thrown out within its substance or upon its free surface. However, it is questionable whether, strictly speaking, all these forms of disease ought to be regarded as inflammatory, as, in my opinion, an interstitial exudation is neither a necessary condition nor a sure criterion of inflammation. We shall not, however, indulge in further discussion of the question, and, after the example of *Simon*, in the following chapters, shall employ the term dermatitis to signify a process attended by exudation.

Infiltration of a somewhat extensive portion of skin results in the so-called erythematous and erysipelatous inflammations. When the infiltration is limited to small discrete spots of the capillary layer, we have the papular exanthemata. The variety of dermatitis, characterized by the formation of wheals or wales, proceeds from a more superficial and extended infiltration. In the erysipelatous form of inflammation, a simultaneous exudation upon the free surface of the cutis often lifts the epidermis, forming vesicles and blebs of variable size. An exudation upon the surface of the cutis, which elevates the epidermis into blisters, also occurs in other kinds of inflammation, both acute and chronic, which are not attended by infiltration of the skin itself.

The vesicular exanthemata consist in superficial inflammation which, like catarrh, is characterized by exudation upon the free surface of the skin, unaccompanied by serious organic lesion, while in the pustular form the exudation contains an abundant admixture of young cells. There are also forms of cutaneous inflammation where the disease of the cutis is accompanied by a morbidly profuse growth of epidermis, the so-called *psoriasis*. Besides these varieties, which differ chiefly in the intensity of the inflammation and the seat of the exudation, there are others distinguished by the peculiar character of the course of the disease, and by the causes which produce it, and which compel us to treat of them in separate chapters, in order to avoid the classification under one head of dissimilar diseases.

CHAPTER III.

THE SLIGHTER FORM OF ACUTE INFLAMMATION OF THE SKIN WITHOUT VESICATION—ERYTHEMATOUS DERMATITIS—ERYTHEMA.

Etiology.—In this form of dermatitis, the papillary layer of the skin, and, in most cases, the tissue of the cutis, becomes the seat of hyperæmia and of serous infiltration. Since the epidermis usually scales off after the erythema has subsided, it is to be presumed that the attachment of the cuticle to the papillary layer has been loosened by a simultaneous effusion upon the surface of the cutis. This effusion, however, is insufficient in quantity to raise the epidermis into blisters. Heat, the direct rays of the sun, mechanical and chemical irritation, and other exciting causes to which we already have alluded in speaking of hyperæmia of the skin, when allowed to act with greater intensity and for a longer time, give rise to erythematous inflammation. Special names have been given to some forms of erythema from mechanical irritation. Where it arises from the friction of two opposing surfaces, it is called *erythema intertrigo*. This is very common among infants, especially among feeble ones, appearing in the folds of the skin, upon the genitals, behind the ears, and in the neck. It also occurs underneath the pendulous breasts of very corpulent females. When it arises between the nates, from their continuous friction upon one another during a long march, it is popularly called "*a wolf*" ("Wolf"). The erythema which appears in cases of protracted illness, caused by the pressure of the bed upon the sacrum, trochanters, and other prominent parts of the body, is called *decubitus*. That which is induced by tension of the skin in cases of severe dropsy, or by trifling wounds, takes the name of *erythema lœve*. Where there long has been a discharge from the nose, and in blenorrhœa of the conjunctiva, the acrid secretion which constantly bathes the skin often gives rise to a superficial erythema of the upper lip and nose. Erythema of the prepuce, scrotum, and inner surfaces of the thighs also results from the continual moisture of the skin in cases of incontinence of urine.

Besides these forms of erythema, which are the result of local irritation, there is another variety, the symptoms and course of which are peculiar, and the cause of which is unknown. In this country it sometimes appears sporadically, but not often, but shows a great tendency to periodic relapses. In other places (Constantinople, Paris), it has been observed to extend epidemically.

Symptoms and Course.—Erythema, arising from local irritation, is characterized by red, slightly-prominent patches, the color of which grad-

ually becomes fainter at the edges. They become pale when pressed upon, assuming a yellowish tinge; and, when the pressure is removed, the redness returns. The spots are the seat of a more or less severe pain. If the irritant which has caused the erythema be speedily removed, the redness and swelling disappear in a day or two, and a slight desquamation of the epidermis finishes the process, which is always an insignificant one. If the irritant be not removed; if it be allowed to operate continuously and vigorously, the erythema assumes a more serious character, and new lesions form. *Erythema solare* becomes an *eczema solare;* blisters form upon burns; in intertrigo, the epidermis is lost; while erythema læve may terminate in gangrene of the skin.

Spontaneous erythema, that is to say, the erythema which appears without assignable cause, always attacks the dorsal surface of the hands and feet, much more rarely the body or face, the backs of the hands and feet always suffering at the same time. *Hebra* lays stress upon the constancy in the situation of the disease as an important diagnostic point. The attack commences with a moderate reddening and tumefaction of the regions just mentioned as its first symptom. Soon nodules and lumps of a deeper red hue, and sometimes even of a bluish red, appear upon the reddened and swollen basis. Hence the name erythema papulatum seu tuberculosum. In some persons this eruption is attended by a disagreeable sense of burning at the point affected; in others there is fever. In a day or two the redness and swelling about the nodules begin to subside; a day or two later the lumps themselves grow smaller and paler, and finally disappear altogether. The epidermis scales off, and the entire duration of the disease is from one to two weeks. Both the bluish color of the nodules, and the yellowish tinge of the skin where the nodules were situated, which remains for some time after the nodules have disappeared, show that, besides the exudation, erythema papulatum is also attended by slight extravasations of blood into the tissue of the cutis. Sometimes erythema papulatum becomes chronic, and lasts for weeks and months; it then spreads from the point first attacked to other regions. When new nodules form upon the periphery of the first eruption, the disease meantime having abated in its centre, it is called erythema annulare or circinatum. If there be a red spot remaining in the middle of the ring, it is called erythema iris, or mamillatum. If, as the circles extend, they become confluent and broken at the point of contact, and thus form arched lines, they constitute the so-called erythema gyratum. These names recur in other eruptions, in which the disease extends upon the periphery of the original seat of the eruption, having subsided at the centre, that is, at the point first attacked. The various

lines or marks which are thus made upon the skin are not indicative of different forms of disease, but merely of different stages of development of the same morbid process.

Erythema papulatum or tuberculosum, however, differs from *erythema nodosum*. This affection is most liable to attack young persons, and is far more common in females than in males. Its seat is almost without exception upon the lower extremities, especially upon the legs. It consists in small circumscribed infiltrations in the deeper layers of the skin, accompanied by extravasation of blood. We at first perceive rounded knots of the size of a hazel-nut or of a walnut, covered by slightly-reddened skin, and somewhat painful to the touch, so that they strongly resemble bruises (dermatitis contusiformis). The rose-red color of the skin gradually grows darker, and then changes successively to a violet, to blue, green, and finally to yellow, the series of changes being entirely similar to that which occurs in traumatic extravasations beneath the skin. Erythema nodosum is always accompanied by febrile disturbance, which debilitates the patient and confines him to bed. The disease usually lasts one or two weeks, and its termination is also followed by desquamation of the epidermis. As a great rarity, erythema nodosum also becomes chronic, the first nodes subsiding with desquamation of the epidermis, and new ones succeeding them.[1]

Treatment.—Erythema resulting from local irritation soon subsides when the exciting cause is removed. When the burning pain is severe, we may make applications of cold water or of lead water. In erythema intertrigo, in order to prevent the friction of the opposing surfaces, we must sprinkle them with fine powder, lycopodium seeds mixed with oxide of zinc being the one most in use (sem. lycopod. ℥ ss, zinci oxid. ʒ ss), or else a pledget of charpie, smeared with zinc ointment, may be inserted between the surfaces. Erythema arising from the pressure against the bed (*decubitus*) may be relieved by the use of circular india-rubber pads filled with air. When it results from contact of acrid secretion, the skin should be protected from it by a coating of lip-salve or other grease. Erythema papulatum requires no particular treatment, although, if it be attended by much burning, we may make use of cold compresses. In erythema nodosum proper attention must be paid to the fever and to the strength of the patient. Compresses wet with cold water, or with lead-water, should be applied to the nodules if they are painful.

CHAPTER IV.

ERYSIPELATOUS DERMATITIS—ERYSIPELAS.

Etiology.—Erysipelatous dermatitis is distinguished by an intense hyperæmia of the cutis, and by a profuse serous transudation, not only into the skin itself, but into the subcutaneous areolar tissue, and sometimes between the cutis and the epidermis. It is also marked by the slightness of its tendency to form abscesses, and by the invariable implication of the lymphatic vessels and glands in the inflammation. Erysipelas not unfrequently results in rupture of small vessels and of hæmorrhages into the skin and upon its free surface. The disease is sometimes so violent as to end in gangrene.

I believe that it is an error to suppose that any cutaneous irritant, if of sufficient intensity, may produce erysipelas. Comparison of erysipelatous inflammation with that caused by a blistering plaster, in spite of the blisters which may form in either case, seems to me decidedly to contradict the identity of the two processes. Besides, the inflammatory derangements of nutrition produced by burns and by mechanical injuries, and similar sources of local irritation, do not present the characteristics of erysipelas, as they either produce simple blisters without any other exudation either in the skin or under it, or else, when of greater intensity, cause destructive disease of the part. We may regard it as proved that most forms of erysipelatous dermatitis proceed from the extension of an inflammation from the wall of an inflamed lymphatic vessel into the surrounding tissue of the cutis. First among these forms is that resulting from inoculation with some acrid or venomous material. In such cases it can often be demonstrated by direct observation that the poison was first taken up by the lymphatics, and thus gave rise to inflammation of their walls, and that the inflammation of the skin is a secondary occurrence. Here a reddened cord, with nodular enlargements, is first observed, and it is not until afterward that a diffuse and uniform redness and swelling make their appearance upon the skin. Next to this form comes the erysipelas, arising from the absorption of the ichorous secretion of a wound, or of the putrid contents of an abscess by the lymphatics, and their consequent inflammation. Not to encroach too far upon the province of surgery, I will merely call to mind the so-called "tooth-rose." This consists in a dermatitis with all the peculiarities of erysipelas; and there can hardly be any doubt that it proceeds from the extension of a lymphangitis to the skin, and that it is caused by the absorption of the liquid from the fœtid contents of a gumboil. The simplest explanation, for the fact that, in hospitals, erysipelas now

and then attacks the most trifling wounds, even leech-bites, is, that the material which excites inflammation in the lymphatics, when absorbed by them, needs not absolutely to be inoculated, nor to have been generated by the decomposition of the secretion of a wound, but that it may be contained in the surrounding air or in the dressings which cover the wound. Although, however, I regard it as almost certain that many forms of erysipelas proceed from the extension of an inflammation from the walls of the lymphatics to the skin, yet I do not believe that all forms of the disease arise in this way; and, in the so-called *erysipelas verum seu exanthematicum* in particular, I believe this mode of origin to be improbable. This primary idiopathic form of dermatitis, which attacks persons previously in good health, and which appears most commonly upon the face and scalp, bears some analogy to pneumonia, pleurisy, laryngitis, bronchitis, angina, etc., which also occur primarily and idiopathically, and which likewise appears in previously healthy subjects. We have no reason for looking upon true erysipelas as one of the acute exanthemata, or of regarding it as infectious; indeed, the great tendency which the complaint shows to recur again and again, in the same individual, may be advanced as an argument against its analogy with scarlatina, small-pox, measles, etc., and against the supposition of its infectious origin. The causes of exanthematic erysipelas are as obscure as are those of pneumonia and the other inflammations above alluded to. In both forms of disease it cannot generally be ascribed to local irritation, nor to cold, to errors of diet, nor to other pernicious agents. Violent mental emotion, however, seems to have an influence in producing the disease, especially in persons who already have suffered from it. Like pneumonia, angina, and the other inflammations, erysipelas sometimes prevails without assignable cause, under the influence of a so-called genius epidemicus stationarius. We are unable to determine whether these inflammatory disorders are secondary local manifestations of some primary general disease, or whether the affection is of a local character from the outset. Erysipelas also resembles pneumonia and the other inflammations, by its tendency to attack persons who have already suffered from the disease in preference to those who never have had it. It occurs most often during middle age, and is seen somewhat more frequently in women than in men. The disease is more common in warm weather than in cold.

Symptoms and Course.—Erysipelas proceeding from external causes belongs to the province of surgery. In this work we shall only consider the symptoms and course of the *erysipelas verum seu exanthematicum.*

In many cases of true erysipelas, the local symptoms are preceded

by some hours, and still more often by some days, of general disturbance of the health, accompanied by more or less fever. As the prodromic stage is not constant, and as sometimes the general derangement of health and the fever do not appear until after the local symptoms, the precursory signs of erysipelas are not to be compared with the fever of the acute exanthemata, but are rather to be regarded as analogous to that which often, for several hours or days, precedes a severe coryza, or the stitch and cough of a pneumonia. The first local symptom of the disease which is developing, is a sense of heat, tension, and pain in the skin, which is not as yet reddened or swollen The neighboring lymphatic glands are already enlarged and sensitive to the touch. Soon the skin, begins to redden and to swell. At first the redness is speckled and clear, but it soon becomes diffuse and dark. The swelling increases and soon becomes extreme, especially where the skin is attached to the parts beneath by loose connective tissue (as in the eyelids), where it renders the skin smooth and shining from the tension. With the swelling, the sense of burning and fulness also increases. At this period there is almost always a violent fever, which grows worse toward evening. The pulse is usually full, beating from a hundred to a hundred and twenty times a minute, and the temperature rises to 105° F., or even higher. The thirst is increased, the appetite lost. As in facial erysipelas, the mucous membranes of the mouth and tongue sympathize with the inflammation of the skin; there are also signs of violent oral catarrh. The tongue is heavily coated and dry from the effects of fever, while a fetid odor proceeds from the putrefying epithelium which covers it. There is a slimy or bitter taste in the mouth.

Although facial erysipelas may also be accompanied by symptoms of dyspepsia, yet, from this, we are not justified in assuming that the disease proceeds from a saburral condition, or from "biliousness," as nearly all fevers are accompanied by more or less gastric derangement, the patient sleeps badly, and his rest is troubled by dreams. Sometimes there is delirium, which, however, but rarely depends upon implication of the meninges, and is due to the fever alone. At this stage of facial erysipelas the patient is much disfigured and scarcely recognizable. The epidermis is usually elevated here and there in small vesicles, or even in large blisters, while elsewhere the blisters have burst, and their contents, with the débris of the epidermis, are dried into yellow scabs. It is quite useless to give special names to the unimportant variations observed in different cases of erysipelas, caused by the degree of the swelling, the presence or absence of vesication, the size of the blisters, the nature of their contents, such as *erysipelas lævigatum s. erythematosum,—miliare, vesiculosum, bullo*

sum, *crustosum*, and the like. The redness usually begins to fade about the third or fourth day, the swelling subsides, the pain abates, and the tension of the blisters, which still remain, diminishes, part of their contents undergoing absorption, another portion drying up into a crust. It nearly always happens, in facial erysipelas, that the disease spreads beyond the region first attacked, and that while the inflammation is subsiding at one point it is still at its height elsewhere. Hence, patients often suffer more after the tumefaction of the face has subsided, and they have again become able to open their eyes, because the disease has invaded the scalp, which, being firmly attached and but slightly distensible, is much more sensitive when in a state of inflammatory tension.

Excepting the "wandering erysipelas," to be described presently, the disease does not generally spread over more than a moderate area of skin. Facial erysipelas almost always involves the eyes, ears, hairy scalp, and a portion of the throat, but scarcely ever attacks the back of the neck or the trunk. Hence, as it also has to run its course upon the region last attacked, its whole duration is usually about a week, or somewhat longer. The process terminates in the peeling off of the cuticle in large pieces, even where there were no blisters. If the scalp has been inflamed, the hair always falls out soon afterward, for the hair follicles have also been the seat of an exudation which loosens the hair and detaches it from its matrix. Erysipelas, however, does not cause any permanent injury of the hair follicles, hence the baldness which it occasions soon disappears completely. In the somewhat rare instances in which erysipelatous dermatitis passes into suppuration, fluctuation appears at one or more points of no great magnitude (and most frequently in the eyelids). Generally speaking, this is not observed until the redness and swelling of the surrounding parts have begun to subside. Puncture of the abscess or its spontaneous opening, which is apt to be somwhat delayed, is usually followed by a discharge of yellow laudable pus, and by speedy recovery. Reddening of the contents of the vesicles by effusion of blood is not always an unfavorable sign, although it is sometimes due to a gangrenous stasis of the capillary circulation in the inflamed skin. Upon the appearance of gangrene of the skin (which is not rare in the malignant forms of symptomatic erysipelas, but which is of quite exceptional occurrence in the true, or exanthematic form of the disease), the contents of the vesicles assume a dark color, and the skin beneath is converted into a grayish discolored slough. The general condition of the patient changes. The fever becomes asthenic; the temperature is very high; the pulse very small and frequent, and there is an intense prostration which may be fatal to life. Even the most favorable cases of gan

grenous erysipelas are very tedious in their course, as the loss of substance resulting from the separation of the sloughs is repaired very slowly.

Erysipelas is sometimes complicated with bronchial or with intestinal catarrh, sometimes with intense hyperæmia of the kidneys, and with catarrhal or croupous inflammation of the uriniferous tubules. A far more serious but less common complication is the extension of the inflammation from the scalp to the meninges. There is no metastasis in such cases, however. The so-called "striking-in" of the erysipelas is a consequence of the malignant character of the disease, and of incipient collapse, and is not to be regarded as the cause.

Wandering erysipelas (*erysipelas ambulans s. migrans*) generally attacks the extremities, and spreads toward the trunk and head. It usually advances steadily, so that the disease, while subsiding in one place, is commencing in the immediately adjoining one. Much more rarely it advances by skips, so that there are intervening regions of sound skin between the inflamed surfaces. The hyperæmia and swelling are not generally so severe in the wandering erysipelas as in the stationary; and while in the latter the redness and tumefaction are most intense at the centre and gradually fade toward the edges, in the former the seat of the greatest swelling and reddening is close to the healthy skin. This form of erysipelas is usually accompanied by a more moderate fever. However, as the disease often continues for weeks and even for months, during which time the inflammation sometimes reverses the direction of its progress, and even goes back to its original point of departure, even this lesser degree of fever is capable of so exhausting the patient as to endanger life.

Treatment.—A large number of so-called "sympathetic remedies" against "the rose" are in popular use; even intelligent and educated people often carry amulets to protect them from its attacks, and submit to conjuration of the disease if the amulet fail. Owing to the short, cyclical, and almost invariably favorable course of the disease, therefore, every new attack of erysipelas is a fresh proof, in the eyes of superstitious people, of the efficacy of their sympathetic cures. Hence, as one seldom receives any thanks for attempting to combat such superstition, and as it usually is labor lost; moreover, as the "invocation of the rose" has this advantage, that the patients, confident in the efficacy of the procedure, accommodate themselves to their distressing position with patience, and without longing for superfluous medication, I deem it advisable to allow patients, who believe in such remedies, to do as they like. At all events, they do much better thus than when (as often happens) the physician commences the treatment of

every erysipelas with an emetic, because the tongue is coated and the breath is foul, and when he attacks the inflammation with all the external and internal antiphlogistics, and applies irritants to the already inflamed skin, in order to prevent the "striking-in" of the disease. However, although in most cases we cannot cut the disease short, and as it almost always terminates favorably, even without treatment, it is better to let it alone, or, at most, to envelop the inflamed part in cotton; still accidents may arise, which call for active interference. When the skin is very tense and painful, *Skoda* strongly recommends the application of cold in the form of wet or iced compresses. But existing prejudices will oppose insuperable obstacles to applications of this kind, and, as any accident which may occur under such treatment will certainly be attributed by the laity to repression of the disease, I prefer, not so much on my own account, but to save the patient and his relatives from useless and erroneous qualms of conscience, to resort to slight compression and to mild scarification instead of cold. The result is the same. The painful tension usually soon subsides under the compression induced by painting the inflamed surface with collodion, similar relief is obtained by making minute superficial punctures with the point of a lancet. One or two pencillings of the inflamed surface and the surrounding parts with lunar-caustic in substance, or painting it with a somewhat strong solution of nitrate of silver (argent. nitr. ℈iv, acid. nitric. gtt. viij; aquæ dest. ℥ ss), seems to have a similar action, although I cannot speak from my own experience. The practice, formerly much in vogue, of drawing a line with lunar-caustic around the area of inflammation, in order to prevent its spreading, has proved useless, and is now generally abandoned. Especial attention should be paid to the fever in treating erysipelas, particularly to that slow form of moderate but very persistent fever which accompanies erysipelas ambulans. Quinia and its preparations are particularly appropriate to such cases, as are also a nutritious diet, wine and strong-beer.

Willan's indorsement of *William's* treatment of erysipelas with from four to eight ounces of port-wine daily, and his assertion that the worst case of erysipelas of the head and scalp that he ever saw was cured by Burton ale, should be accepted as meaning merely, that when the fever threatens to consume the patient, alcoholic stimulants must be given freely. The complications of the disease, especially the meningitis, are to be treated upon principles already laid down. When the inflammation disappears, it is not advisable to attempt to reëstablish it upon the surface again by means of vesicants and other cutaneous irritants. Abscesses and gangrene of the skin are to be treated upon surgical principles.

The treatment of phlegmonous erysipelas, and of boils and carbuncles, we refer to the handbooks of surgery.

CHAPTER V.

HERPES—ACUTE SUPERFICIAL DERMATITIS, ATTENDED BY FORMATION OF GROUPS OF VESICLES UPON THE SKIN.

Etiology.—Herpes closely resembles erysipelas, in being an acute dermatitis arising from unknown causes. It differs from it, however, both in the abruptness of the limits of the inflammation and in its seat, which is confined exclusively to the most superficial layers of the skin. *Hebra* defines the various forms of herpes as "a series of acute cutaneous diseases of cyclical course, marked by an exudation which collects in drops under the epidermis and elevates it; forming vesicles which are never solitary but always appear in groups." These vesicles are all of about the same size and shape. The various groups do not appear simultaneously, but follow one another at intervals of several days, so that crops of recent vesicles and of older declining ones are usually to be found at the same time. Eruptions of herpetic vesicles (called *hydroa febriles*) often appear upon the face, and especially upon the lips of patients suffering from pneumonia, intermittent fever, ephemera, and epidemic cerebro-spinal meningitis. This eruption is hardly ever seen in other diseases, particularly in typhoid fever (typhus abdominalis). The appearance of herpes upon the face in acute febrile disease has long been regarded as a favorable prognostic sign; perhaps merely because the diseases, to which it is almost peculiar, recover more frequently than those in which it is rarely or never seen. Herpetic eruptions, however, occur also in individuals previously healthy, as well as during the course of other diseases.

It may be regarded as established that *herpes zoster* depends upon disease of the trophical fibres of the motor and sensory nerves, which supply the part affected. Further confirmation is required of the supposition that all forms of herpes are of similar origin; for instance, that herpes labialis proceeds from disease of small nervous branches in the lips.

Symptoms and Course.—According to the region attacked, herpes is classified into, first, *herpes labialis*, which is situated upon the lips, and which often extends to the mucous membrane of the mouth. When the groups of vesicles appear upon some other part of the face, such as the cheeks or eyelids, it is called *herpes phlyctenodes*. *Hebra* proposes the name of *herpes facialis* for all forms of herpes appearing upon the face. There is also a *herpes preputialis*, which, as it likewise

arises upon other parts of the external genitals, might more properly be called herpes *pudendalis*. Finally, there is herpes *zoster* or *zona* (shingles), which extends, in a very peculiar manner, along the course of the cutaneous nerves. When herpes zoster appears upon the thorax, the groups of vesicles form a somewhat broad, interrupted belt, which begins at one of the vertebræ, and, following the line of one of the intercostal spaces, reaches to the sternum, but scarcely ever occurs upon both sides of the body. When the eruption is upon the belly, the groups of vesicles are arranged much as they are in zoster of the chest, and extend from the lumbar vertebræ to the linea alba and mons veneris. Upon the neck the vesicles sometimes form a half-collar, sometimes they extend downward toward the second rib. Upon the face, the eruption spreads along the course of the facial nerve, especially along the cheek, to the dorsum of the nose. Upon the scalp it runs over the forehead and skull along the course of the supraorbital nerve, or else spreads over the occiput along the course of the occipital nerve. Finally, in zoster of the arm and thigh, the eruption of vesicles follows the courses of the nerves which spring from the brachial and crural plexus. All forms of herpes begin with a sense of burning pain, usually not of a very severe character, in the affected region. Numerous red points soon become visible, which coalesce, forming red specks of irregular shape, which, on the next day, usually are covered with small transparent vesicles. The contents of the vesicles, which rarely exceed the size of a lentil or a split pea, become turbid in two or three days, or else reddened from admixture of blood. About the third or fourth day the vesicles commence to shrivel, and they and their contents subsequently dry up into a brownish scab. The scabs fall off in from ten days to a fortnight after the first appearance of the eruption, and for some time afterward there remains a reddish spot, covered with thin epidermis. The pain, which is of a burning character, though not very severe, usually abates when the vesicles begin to shrivel. Herpes zoster is sometimes accompanied by fever, and in rare instances the eruption of vesicles is preceded by febrile disturbance, like the inflammatory cutaneous exudation of erysipelas. There is no fever in the other varieties of herpes, or the fever, if present, does not depend upon the herpes, but upon the disease with which the herpes is associated.

Besides the forms of herpes above enumerated, there is also a *herpes circinatus* and a *herpes iris*. These species are not classified according to their locality, like the other forms, but according to the arrangement of the vesicles. This is quite analogous to that of the nodules in erythema circinatum and erythema iris. In herpes circinatus, a circle of vesicles encloses a tract of healthy skin. Moreover, the vesicles themselves are usually smaller than those of other kinds of

herpes, and they do not always form a scab as they subside, but terminate in reabsorption of their contents, with desquamation. In herpes iris a few solitary vesicles stand in the centre of a circle of vesicles, or of several concentric circles of them, whose vesicles exhibit different stages of development and decline. Most cases both of herpes circinatus and herpes iris, if not all of them, owe their origin to the presence of vegetable parasites.

Treatment.—As the various forms of herpes run a cyclical course, and as they soon subside, and are not followed by any serious consequences, their treatment should be simply expectant. It is sufficient to protect the vesicles, and the scabs after they have formed, from friction or other violence. In herpes zoster, which is the most exposed to friction, the eruption should be enveloped in cotton, which should be allowed to remain just as in the treatment of slight burns. The expectant treatment is especially advisable in herpes preputialis. The spontaneous healing of an excoriation, which remains after the bursting of a vesicle, affords the best confirmation of the diagnosis that we have to deal with a herpes and not with a chancre. True, the diagnosis is easy enough at first, as the grouping of the vesicles in herpes is quite characteristic, and, in the excoriation which remains immediately after they have burst, it is easy to perceive from its shape that it has been formed by a cluster of several vesicles. Somewhat later, however (especially if the patient, in his anxiety, have cauterized them with lunar-caustic), the diagnosis is more difficult, and frequently our only sure criterion is in observation of their subsequent progress. If the excoriation heal in a few days, under the application, twice a day, of a bit of lint moistened in water, between the prepuce and glans, or if the recovery is not delayed beyond a week, we may be sure that it is not a chancre.

CHAPTER VI.

URTICARIA—NETTLE-RASH — ACUTE SUPERFICIAL DERMATITIS, WITH FORMATION OF WEALS.

Etiology.—In urticaria a serous infiltration of the papillæ of the skin, and, probably, too, a swelling and infiltration of the cells of the rete Malpighii cause the formation of circumscribed flattened elevations, whose width is greater than their height, and which are called weals. Owing to the rapidity with which the infiltration that produces the weals appears and disappears, we should not have reckoned urticaria among the inflammatory affections, but should rather have described it as a local œdema characterized by great peculiarities in its

distribution and limits, were it not that in a previous section we have applied the title dermatitis to all the exudative diseases of the skin without considering whether or not they are accompanied by genuine inflammatory symptoms. We have also placed urticaria among the acute inflammations, although there are cases in which patients have suffered for years from it. However, the disease has not been a chronic one in such cases, but rather a series of acute relapses occurring at very short intervals. The causes of nettle-rash are very varied. Some of them we know, the rest are quite unknown. Urticaria has been classified according to the different exciting agents which produce it.

1. *Urticaria from External Irritation.*—Under this head come all forms of nettle-rash arising from local irritation of the skin from contact with stinging nettles, with the leaves of the rhus toxicodendron, with the hairs of certain caterpillars, and with some of the mollusca. It also includes the rash induced by the bites of fleas and midges, and that caused in the skin of some people by scratching with the finger-nails.

2. *Urticaria from Ingesta.*—This variety breaks out upon some persons immediately after they have eaten strawberries, crabs, muscles, mushrooms, or other unaccustomed food. It is quite hypothetical to assume that in these cases an acrid material enters the blood and upon reaching the skin occasions the irritation. It is very strange that such food should only have this effect upon a few people, and that in these it never fails to produce urticaria. The rash which we not unfrequently see after the exhibition of large doses of balsam copaiba is also included among the urticaria *ab ingestis.*

3. *Urticaria febrilis* or *febris urticata.*—The causes of this form of the disease, which is accompanied by severe fever and gastric derangement, and which greatly resembles the acute exanthemata in its course, are unknown.

4. *Chronic Urticaria.*—The cause of this somewhat rare disease is also obscure. It occasionally seems to depend upon hereditary predisposition.

5. There is a form of urticaria mentioned by *Hebra*, which is evidently dependent upon uterine irritation, and which appears in some women during pregnancy; in others, during menstruation; while in others, again, it accompanies diseases of the womb, or follows the introduction of pessaries.

Symptoms and Course.—The weals always rise from a base reddened by hyperæmia. They themselves, however, are often white (*urticaria alba s. porcellana*), probably owing to compression of the vessels of the papillæ by the infiltration. Sometimes they are dis

crete; at others, they stand so closely together as to coalesce (*urticaria conferta*). Sometimes the duration of the individual weals is very brief (*u. evanida*), then again it may be very persistent (*u. perstans*). When the weals are large and hard, the urticaria is called *tuberous;* when small, it is said to be *papulous* (*lichen urticarius*); and, if the epidermis upon them be elevated into the vesicles here and there, it is called urticaria *vesiculosa*. The rash is always accompanied by a most tormenting itching of the part, so that the patient cannot refrain from scratching it. With exception of the febrile urticaria, the itching and the objective signs upon the skin are the only symptoms of all forms of the disease. It seldom lasts over a few days; although, in chronic urticaria, the rash recurs at short intervals for weeks, months, and years, one eruption following another, but none of them ever lasting very long. The fever which accompanies and sometimes precedes the febrile urticaria may be so intense as to cause dryness of the tongue, disturbed sleep, and even delirium. If the fever be attended by violent vomiting (and, as often happens, by diarrhœa, due, no doubt, to an affection of the intestinal mucous membrane similar to that upon the skin), the disease may present an aspect of great gravity. However, the eruption, fever, and gastric disturbance subside in a few days, and a rapid convalescence ensues.

Treatment.—It would be a great gain, could we only relieve patients from the itching of that form of urticaria which sets in rapidly and subsides spontaneously, and which so torments them as to deprive them of all comfort and sleep. The palliatives recommended for this purpose, however, the chief of which are bathing the skin in very dilute acids, and rubbing it with slices of lemon, are often ineffectual. Neither do we know of any efficient remedy for chronic urticaria. We must, therefore, confine our treatment to the endeavor to restore the general health of the patient by suitable diet and medicines, to correct digestive derangement, and to forbidding the use of such articles of food as we know to be liable to cause the disease.

CHAPTER VII.

ECZEMA—DIFFUSE SUPERFICIAL DERMATITIS, WITH SEROUS EXUDATION UPON THE FREE SURFACE, AND WITHOUT TYPICAL COURSE.

Etiology.—By far the most common form of dermatitis is eczema. As in herpes, the inflammation is limited to the superficial layers of the skin, and is accompanied by a serous exudation upon its free surface. But it differs from herpes, on the one hand, in its tendency to spread over the surface—a tendency which is quite unmistakable even

in eczemas of very small extent; and, on the other hand, it differs in the irregularity of its course, which, unlike that of herpes, is not confined to any fixed period of time. Eczema may be regarded as the analogue of catarrh. It is the most common of all diseases of the skin, just as catarrh is the most common affection of the mucous membranes. Like catarrh, too, it is a disease of the surface rather than of the parenchyma, and it is accompanied by a profuse superficial serous transudation. It also usually involves wide tracts of the skin, or, if its area be small, it shows a tendency to spread.

Eczema being a diffuse dermatitis, with superficial serous exudation, it is plain that the formation of vesicles must be a frequent occurrence in this disease; but it is equally certain that the presence of vesicles is by no means one of its constant or essential symptoms. If the superficial exudation be profuse enough to form small drops, and if the epidermis possess sufficient resisting power, not to give way immediately before it, vesicles form, producing the variety of eczema known as *eczema simplex, seu vesiculosum.* When there is a profuse admixture of young cells with the contents of the vesicles (which always contain a few such cells), so that the serum is turbid, yellow, and purulent, the vesicles become pustules, and the disease is called *eczema impetiginosum.* When the transudation is not so copious as to elevate or to break through the epidermis, it usually soon dries up; and then, instead of vesicles or pustules, nothing is to be seen except dry scales rising from the reddened skin. This has been called *pityriasis rubra,* and is now known as *eczema squamosum.*

From its anatomical and pathological point of view, I cannot regard the form of pityriasis rubra, which *Hebra* considers as distinct from eczema squamosum, as any thing more than a squamous eczema capable of endangering life from its superficial extent. According to *Hebra's* description, this affection is also a superficial dermatitis, whose scanty superficial exudation, mingled with epidermic cells, dries into scales, and which, like all exanthemata involving the whole surface, is a dangerous disease.

Finally, when the exudation detaches the epidermis so as to deprive the red, moist corium of its covering, and expose it to view, it is called *eczema rubrum* ("*salt rheum*"). After the epidermis has disappeared, the exudation upon the surface often dries up into scabs and crusts, whence many other names formerly applied to moist eczema were derived, such as *tineæ* or *crustæ lacteæ, granulatæ, mucosæ,* etc., etc. *Hebra* also counts that form of eruption as an eczema which is usually called *lichen,* and which appears as a rash of solid papules. He thus adds a fifth species, *eczema papulosum,* to the series. Since a serous exudation is also thrown out upon the surface in the papular erup-

tions, which swells up the cells of the rete Malpighii, but is not copious enough to overflow its free surface and to elevate the horny layer of the epidermis into vesicles, we accept the classification of lichen as a form of eczema; but we deem it improper to make exception of certain of its varieties, and to distinguish them from eczema as a separate affection.

The causes of eczema are: first, direct irritation of the skin. The action of an atmosphere hotter than the normal temperature of the body is the cause of eczema caloricum, which, no doubt, is identical with the *calori* of the Italians, and with the lichen tropicus. Eczema *solare* is produced by the rays of the sun. The effect of warm baths of simple or medicated water is to produce a kind of eczema known as "bath itch," while abuse of the skin, by means of cold compresses and cold douches, gives rise to the "critical" eruptions of the hydropaths, and the inunction of blue ointment will cause a mercurial eczema. If we were to give a name to the eczemas which proceed from other causes, such as the action of the vegetable and mineral irritants, parasites, pressure, and the like, the number of species of eczema might be much increased. The itch, a dermatitis usually of an eczematous character, and excited by the presence of the *acarus scabiei* shall be treated of in a separate chapter. *Miliaria rubra*, induced by excessive sweating, is also an eczematous affection. The contents of its vesicles is an inflammatory exudation of an alkaline reaction, and is not acid like the sweat of the *miliaria alba*.

Secondly: Eczema may arise from obstruction of the venous circulation. As such derangements of circulation usually arise in the lower extremities, it is upon these that the eczematous inflammation usually appears and forms an analogue with the catarrh of the stomach, which arises in cases of compression of the portal vein, and with catarrh of the rectum when the hæmorrhoidal vein is obstructed.

Thirdly: Eczema is often of constitutional origin. A tendency to eczema and to other diseases of the skin is often called a herpetic diathesis, or a herpetic dyscrasia. The term herpetic dyscrasia is based upon the idea that the blood and humors of a patient with constitutional eczema have undergone some qualitative or quantitative change. The soundness of such an hypothesis is not only unproved but quite unlikely. Eczema of an indubitably constitutional origin is seen as often in plethoric subjects as in anæmic ones, and in cachectic persons quite as frequently as in those who are robust and whose humors certainly have not deteriorated. No objection can be made to the term herpetic diathesis, because it leaves the question undecided whether the predisposition to eczema and to other cutaneous diseases is dependent upon a perversion of nutrition or upon other conditions. The her

petic diathesis is frequently congenital, and, as *Veil* in Canstatt has demonstrated, is hereditary in many instances. One would naturally suspect congenital predisposition to eczema in a family in which all the brothers and sisters were afflicted by it, and that it was hereditary if the parents and grandparents also have had it. We need hardly state that a herpetic diathesis is not always transmitted from parent to child, any more than any other hereditary disease. Scrofulous and rachitic children always exhibit a most pronounced tendency to eczema, especially to eczema impetiginosum. Sometimes there is a connection between eczema and chronic dyspepsia or derangement of menstruation. In such cases, although we may succeed in healing the eczema by local treatment, yet there will always be relapses of the eruption so long as the other complaint remains uncured. Much more frequently, although there may be a most decided herpetic diathesis, it is not accompanied by general nutritive derangement, nor by disease of any particular organ. The appearance of eczema, unprovoked by any appreciable irritation of the skin, and its frequent recurrence, in spite of the most careful avoidance of all sources of irritation, is often the sole sign of constitutional derangement.

Symptoms and Course.—The subjective signs of eczema consist in itching, accompanied by an irresistible inclination to scratch, symptoms which are common to all cutaneous diseases, where the papillary layer is affected. The objective signs have already been given above. We have already seen that the cutaneous surface, which is the seat of that atypical, diffuse, superficial dermatitis called eczema, sometimes is studded with vesicles; at others, with vesicles and pustules intermingled, and sometimes again with scales; that, in other instances, it presents a red, moist surface, denuded of its epidermis, and that, in still others, it is covered with crusts and scabs.

Besides the four forms of eczema (e. simplex, e. squamosum, e. rubrum, e. impetiginosum), it is also customary to recognize a chronic form of the disease. But not only is it illogical to associate one species, distinguished by the nature of its course, with four others, classified upon an entirely different basis, but in practice this arrangement is found to be attended by many inconveniences. Chronic eczema can only be contrasted with acute eczema; like the latter, it sometimes assumes the simple form, sometimes the squamous or the impetiginous form, and sometimes again that of eczema rubrum. In cases of very long standing, which usually assume the type of eczema rubrum, the disease upon the surface is occasionally accompanied by changes in the structure of the cutis. This generally consists in an inflammatory hypertrophy, quite analogous to that which occurs in the mucous membranes in chronic, bronchial, and gastric catarrh. Less frequently there

is an atrophy of the cutis caused by pressure of the crusts and scabs adhering to it.

Classification of eczema, according to the extent and locality of the surface which it involves, is of much greater importance than that based upon its form and duration. Upon this basis, in the first place, it may be divided into *universal* and *partial* eczema. The term universal, however, is not to be accepted literally, because, although the eruption often involves a very large portion of the surface, it seldom covers the whole of it. Universal eczema is a much less common affection than partial eczema. It is either acute, when it usually assumes the simple or squamous character less frequently than that of eczema rubrum; or else it is a chronic disease, and then takes on different aspects upon different parts of the body, showing preference, however, to the type of eczema rubrum, so that the surface is generally either moist and raw, or else covered with large crusts. Chronic universal eczema is always a very distressing and very obstinate disease, although it does not imperil life, and indeed often does not seem materially to prejudice the health of the patient.

Partial eczema is often situated upon the scalp. Even when the disease commences as a vesicular eruption, the vesicles are apt to be overlooked, and to be destroyed by combing or scratching. If eczema impetiginodes, or E. rubrum, appear on the scalp, the eruption discharges very freely, the hairs are glued together and crusts form, which are sometimes soft and flat, sometimes thick and hard, and to this the name of tinea favosa, granulata, etc., was formerly applied. Such a scalp, covered with scabs, is a very favorite and favorable abode for lice. The cervical glands often become enlarged in moist eczema of the head, and sometimes even suppurate. The disease presents a very different appearance when the transudation is too scanty to form vesicles or to burst through the skin. In such cases it assumes the squamous type, and a great quantity of small white detached scales of epidermis are found, not merely upon the reddened skin, but among the hairs and upon the collar of the coat. This was formerly called *tinea furfuracea;* or *tinea amiantacea*, when the epidermic scales, mixed with exudation, formed thick, shining layers, like asbestos. All forms of eczema frequently appear upon the face, although the impetiginous and rubrous types are the more prevalent ones during childhood. Although it does not spare other regions, its favorite seat is upon the cheeks and chin, which become reddened and shiny, and after a few vesicles have formed upon them, and burst, they are covered by a clear yellow liquid. This soon dries up, forming yellow crusts. If we remove these before the disease has subsided, we come immediately upon the bare, moist corium, and can find no intervening epidermis. Ecze

ma rubrum, et impetiginosum, of the face used formerly to be called *porrigo larvalis*, *tinea faciei*, *crusta lactea*, *crusta serpiginosa*, etc. It often extends into the external auditory meatus, and still more frequently is complicated with coryza, with ophthalmia, and with enlargement of the submaxillary and cervical glands. In many cases facial eczema is confined to the ears, the eyebrows, the eyelids (especially the commissures), and, above all, to the lips. These points are sometimes studded with vesicles; sometimes stripped of their epidermis and bathed in liquid secretion, or covered by scabs, and sometimes again they are attacked by the squamous forms of the eruption. A very obstinate species of eczema rubrum is also observed about the nipples of nursing women, although it may also attack the nipples of women who are not nursing, and even those of children. The vicinity of the navel is also the seat of a partial eczema which is most common in corpulent persons. A very important variety of the disease is eczema pudendorum. In men, it usually attacks the penis and scrotum; in women, the labia majora. Sometimes it is acute, assuming the simple form; sometimes chronic, when it presents a very moist eczema rubrum. The intolerable itching which accompanies it almost drives the patient to desperation. A similar eruption appears about the anus and upon the perinæum, only its secretion is not so profuse as in eczema pudendorum. Under the name of eczema *marginatum*, *Hebra* describes an eruption which is most common among shoemakers and cavalry soldiers. It commences at the point where the scrotum comes into contact with the thigh, but usually extends; and after a while a similar spot appears symmetrically upon the inner surface of the other thigh. Eczema of the lower extremities is most common upon the legs, where it forms large red patches, which either secrete profusely or else are covered by scabs. This is generally called "salt rheum." When the eruption attacks the flexures of the joints, they become covered by a layer of rough crisp epidermis, mingled with dried exudation, which is apt to crack when the joint is extended, and to form painful fissures. Sometimes a moist eruption also appears on the flexures of the joints. It is remarkable that eczema always attacks the hands and feet simultaneously. When its chief seat is upon the dorsum of the extremity, the eruption takes the simple vesicular form, and may easily be mistaken for scabies. Vesicles are not so apt to form upon the volar surface. More usually there is a hard and somewhat thick crust of dried exudation, mixed with epidermis, upon the reddened cutis, and as this coating of the palms and soles is constantly scaling off in the form of white scales, eczema at these points is often and erroneously called psoriasis, for pityriasis, palmaris, or plantaris.

Treatment.—Errors in the treatment of cutaneous diseases in general, and in that of eczema in particular, are liable to assume one of two directions. One class of physicians are possessed by the prejudice that all local treatment is wrong, because they can never be sure that the suppression of an eruption will not be attended by dangerous consequences. Hence, from the apprehension (which certainly is nearly always groundless) that they may, perhaps, do harm to their patients by treating eruptions locally, they resort to an internal medication, which is injurious beyond all doubt. Another party, blindly following the authority of *Hebra*, look upon all internal medication as superfluous, and treat all cutaneous diseases locally, without the exception even of cases where an eruption has been followed by the abatement of serious disease of internal organs. *Hebra's* success leaves no doubt that the direct treatment of disease of the skin is the best for many patients. Not only is the disorder itself relieved and cured more surely and quickly by such means, but it often does much less harm to the patient than the old-fashioned practice of giving laxatives, metallic preparations, and other noxious medicines. Moreover, with few exceptions, it is a groundless prejudice to suppose that other diseases proceed from the suppression of cutaneous eruptions by means of local applications. On the other hand, it must not be denied that exanthemata, when treated by local means alone, are very apt to relapse; and that cases really do occur now and then, in which there is reason to fear that their repression, under direct applications, may give rise to disease of internal organs.

The forms of eczema in which I consider it inadmissible, or at all events hazardous, to employ vigorous local treatment are—

1. The moist form which appears upon the scalp and face of children. It is an unmistakable fact that the sudden disappearance of such eruptions is often quickly followed by bronchial catarrh, croup, or hydrocephalus; and it is equally certain that a tedious catarrh or other affection will often subside as soon as an eruption of this kind makes its appearance. However, it does not by any means follow that the internal maladies have arisen in consequence of the cessation of the cutaneous disease, nor, on the other hand, have we any proof that it is because of the outbreak of the eruption that they subside. But we are equally uncertain of the contrary propositions, and even the possibility of the existence of such a cause should contraindicate local treatment of moist eczema upon the heads and faces of children. It is true that a similar belief formerly prevailed regarding the supposed injurious effects of the treatment of itch by local means. Such views are now recognized as prejudices, and exploded; and I readily admit that hereafter my fear of treating facial eczema locally may prove equally

groundless; but, according to our present knowledge, such apprehension seems well founded, and the fact, that it is safe to cure other exanthemata by external applications, does not prove any thing regarding the disease in question; since, in spite of the great external similarity of the exanthemata, they differ essentially in their influence upon nutrition, and in their effect upon the general health of the body.

2. Local treatment is contraindicated in all forms of eczema of adults that seem to appear vicariously for other diseases, which have subsided upon the outbreak of the eruption. It is true that *Hebra* expressly affirms that he has suppressed such eczemas without injury to the patient, solely by the employment of local applications; but, in spite of his authority, I should not dare to resort to an external treatment of a case of eczema, the establishment of which had been followed by the recovery of an ophthalmia of long standing, or of a chronic derangement of digestion, or other serious disease.

3. Local treatment, or, at all events, an exclusively local treatment, is unadvisable in an eczema, the cause of which is evidently constitutional. The number of cases suitable for external treatment would then be a very small one, however, in the eyes of the class of physicians who ascribe all exanthemata, whose origin they do not know, to a dyscrasia. One of the important steps recently made in the therapeutics of cutaneous disorders, however, consists in our no longer ascribing an eczema to a constitutional origin, and subjecting it to "anti-dyscratic" treatment, unless we have better evidence of the genuineness of such origin than is afforded by the mere existence of an eruption. The impropriety of treating the syphilitic exanthemata locally is universally admitted, but even in the eczema which afflicts scrofulous and rachitic subjects, as well as in that which attacks chlorotic females, and in that which accompanies disorders of the sexual organs, an exclusively local system of treatment is unwise; not that direct remedies are hurtful in themselves, but they should only be employed as corroborants to general treatment, directed against the fundamental disease. At the same time, we should mention that eruptions which are undoubtedly of constitutional origin often continue, as it were, independently, after the original disease has subsided, and require a vigorous local treatment for their eradication. This is sometimes the case even in syphilitic exanthemata. I knew a merchant in Magdeburg, who, in addition to other signs of syphilis, had a very unsightly eruption upon his face and head, which had lasted for years after all the other syphilitic symptoms had disappeared. This man, after consulting the most eminent physicians, and after undergoing all sorts of anti-syphilitic treatment without benefit, was completely and per-

manently relieved of his disease by means of an ointment of white precipitate and carbonate of lead, prescribed by a second-rate surgeon.

The moist eruptions upon the heads of children, vicarious eczemas, and eczemas of constitutional origin, after all, form but a small class compared with the number of eczemas against whose local treatment no objections can be made, and in the cure of which the most brilliant success may be attained. The topical remedies which I would recommend before all others is the white precipitate in the form of an ointment (hydrarg. ammoniat. ʒ j, adipis. ℥ j), and corrosive sublimate in weak solution (hydrarg. bichlor. gr. j—ij, aquæ destillat. ℥ j), as these articles are much less disagreeable to the patient than tar ointment, soft soap, and the like, and since in a great majority of cases they amply suffice to effect a rapid and complete cure even of the most obstinate forms of eczema. I am quite unable to account for the beneficial action of these articles upon eczematous dermatitis; but I will suggest that both white and red precipitate have long been considered as among the most effective remedies in conjunctivitis. The forms of eczema in which I prefer to use the white precipitate ointment are eczema of the face and scalp, when not too extensive, and particularly when it has not already produced too much thickening of the corium. In such cases the treatment has hardly ever failed me, either in the clinic or in private practice; and under its use I have seen eczemas disappear in a few weeks which had lasted not merely for a year or two, but for eighteen or twenty years. Although I have hesitated to employ it where the eruption was very extensive, from fear of causing mercurial poisoning, yet I have had especial opportunity of satisfying myself that in such cases it likewise does excellent service and does no harm. By means of white precipitate ointment I once cured the wife of an official, in the neighborhood of Greifswald, in a few weeks, of an eczema about the ears and scalp, which had existed for years. Some time afterward, this lady, who was very philanthropic to the poor in her husband's district, and who used to prescribe "homœopathically" for the sick, informed me that she could not thank me sufficiently for my recipe; for she had made a quick and radical cure of a large number of very extensive and obstinate cases of "salt rheum." The patients did not suffer in the least from the rapidity of the cure, and none of them were salivated. The application of white precipitate, however, by no means secures the patient against a relapse, and it is well to warn him of the probability of a fresh outbreak, and to recommence the treatment as soon as he perceives any new signs of the disease. Moreover, as the eczema is all the more easily and quickly cured by the ointment, when the eruption is recent and slight, it is very impor-

tant to enjoin upon the patient to keep a sharp lookout for the first traces of it, as I can illustrate by a most striking example. I cured the wife of an officer, in Magdeburg, in less than a fortnight, of a chronic eczema, for which she had in vain gone through a succession of courses of *Zittman's* decoction, arsenic, iodide of potassium, mercurials, baths, and mineral waters. This eczema had a great tendency to return. When the patient's husband was at home, in Magdeburg, he used every day to examine her face with a lens, and to anoint every suspicious-looking spot with the salve, thus protecting her from any unsightly extension of the disease. When her husband was on duty, and absent from Magdeburg for any length of time, during the autumnal manœuvres of the army, the eczema sometimes spread considerably, and did not afterward yield so readily to the white precipitate, and has sometimes even compelled me to resort to the corrosive sublimate instead. Two or three inunctions daily of this ointment, or an equal number of pencillings with the solution of corrosive sublimate, are usually sufficient. Of course, any adherent scabs must be softened and removed, and the surface must be carefully dried before applying either the ointment or the solution. I have often found that my former pupils, as they came home from their travels, fresh from a course of lectures upon cutaneous diseases, and full of new ideas, at first treat all the eczemas which come to them with soft soap, oil of cade, cod-liver oil, and the like. In a year or two, however, they abandon such treatment, or, at all events, before resorting to it, they try whether the old, simpler, and far more convenient treatment by white precipitate will not answer the purpose. The preparations of lead and zinc stand next in virtue to the white precipitate salve, and the solution of corrosive sublimate, as they are both best adapted for the treatment of spots of eczema of no great magnitude, and where there is no hypertrophic thickening of the subjacent corium. We generally prescribe a solution of sulphate of zinc (zinci sulp. ʒ ss—aquæ ℥ vj), or an ointment of the oxide of zinc, or of the carbonate of lead, in the proportion of a drachm to the ounce of lard; or else, when the patient does not bear salves well, we may order a paste of oxide of zinc with glycerine, or dust the surface with a powder composed of oxide of zinc and lycopodium seeds, or starch (zinci oxid. ʒ j, amyl ℥ j). In the very moist eczema, which arises behind the ears, and in the hollows of the joints, and between the fingers and toes, *Hebra's* diachylon ointment does excellent service. It is to be prepared as follows: Melt the simple diachylon plaster over a slow fire, and add to it equal parts of linseed-oil, and stir the mixture well when cool; or else the following: (℞. olei olivarum optimi ℥ v, lithargyri ʒ x, coque l. a. in molle, dein adde ol. lavendulæ ♁ ij, f. ung. S. Rub the ointment upon the affected spot

two or three times a day, or, what is preferable, apply it upon linen compresses). In very extensive eczema accompanied by great itching, especially in eczema universale, the shower-bath is strongly to be recommended. Some patients are quite unable to tolerate the irritating remedies to be mentioned presently. *Hebra* directs the shower-bath to be used two or three times a day for ten or fifteen minutes at a time in a warm room. By this means the most obstinate eczemas, which have defied all previous treatment, are often made to heal, but only after a somewhat persistent employment of the shower. A suitable apparatus may be made at little cost. In partial eczema, which does not tolerate irritating remedies well, cold compresses may be substituted for the cold douche, especially in recent cases. When the eczema is of very long standing, and particularly when the disease has extended from the surface into the substance of the true skin, and the more difficult it is to pinch up the skin in a fold, so much the more often shall we be compelled to resort to that class of remedies which, from the vigorous alterative action which they exert upon the skin, play an important part in the therapeutics of nearly all inveterate cutaneous affections. Experience teaches that of this class the preparations of sulphur are the least adapted to the treatment of eczema, that it is only in exceptional instances that they do good (as in eczema marginatum, according to *Hebra*), and that in most cases they actually do harm. On the other hand, "green soap," tar, and caustic potash, are of the utmost benefit in old cases of eczema accompanied by infiltration of the corium; hence we shall briefly relate *Hebra's* directions for their employment. The green soap must either be rubbed once or twice daily into the affected surface, or else a piece of flannel is to be smeared with the soap and laid upon the sore spot. This application is also to be renewed once or twice daily, and the procedure should be continued for from three to six days. It must then be discontinued; but the soap which has been rubbed in ought not to be removed. After three days more the patient is to take a bath, when there must be a pause in the treatment. This course is to be repeated until all the infiltration has disappeared, and until the transudation has ceased. At this stage, when a previously moist eruption has changed into a dry scaly one, *Hebra* directs applications of tar to be substituted for the soft soap. Of the various kinds of tar *Hebra* prefers the birch-tar (oleum rusci) and the oil of cade, prepared from the wood of the Juniperus oxycedrus, as having a less offensive smell than the common pine-tar, or oleum empyreumaticum coniferum (pix liquida). Instead of pure tar, or tar-ointment, I for years have used a solution of tar in alcohol (picis liquid., alcohol, āā, or else picis liq., sapon. virid., āā ℥ ss, alcohol ℥ j); for it answers every purpose, is much more con-

venient to apply than pure tar, or than tar-ointment, and is much easier to remove from the skin. The solution is to be rubbed in once a day until the brown coat which forms, instead of being quickly detached, adheres for several days, and until the skin ceases to look red after the brown coat has been shed. As long as the tar-crusts continue to shed rapidly, leaving a reddened surface beneath them, the disease is not extinct. Although the external application of tar is generally borne without inconvenience, yet in some patients the first inunction occasions such an intense irritation of the skin as to render further prosecution of the treatment impracticable. It is still more common for the continued use of tar over a large extent of surface to be followed by violent irritation of the intestine and kidneys, accompanied by vomiting, diarrhœa, the discharge of a blackish-looking urine having a distinct odor of tar, which becomes still plainer upon the addition of one or two drops of sulphuric acid; these symptoms being accompanied by fever and distress in the head, thus necessitating an immediate suspension of the treatment. Not only is the application of soft soap or of tar inadmissible in some cases of eczema, but there are also examples in which, although the remedies are well borne, they do not produce the desired effect. It is in these instances that the cauterization of the part, with a concentrated solution of caustic potash, is indicated (potassæ caustic. ʒ j, aquæ ʒ ij). The cauterization is not to be repeated oftener than once a week. A swab of charpie dipped in the solution is rapidly to be passed over the diseased surface, which is immediately to be covered with cold compresses, in order to allay the intense pain which the application always occasions. Even the most inveterate cases of eczema usually recover after five or six repetitions of the cautery.

Since local applications will merely rid the patient of his eruption, but will not insure him against its return, the question finally arises, whether we should limit ourselves to an exclusively local treatment, or whether the topical applications should be combined with a general internal medication, even when there is no evidence of scrofula or of rachitis, and when the disease is not a consequence of chronic dyspepsia or menstrual derangement. *Hebra* pronounces decidedly against such practice. *Veiel*, on the other hand, who attaches quite as much importance to local treatment as *Hebra* does, combines it with laxative tisans and with iodide of potassium in increasing doses, even when there is no perceptible constitutional taint. The success obtained at *Veiel's* institution is very brilliant; and moreover, it being a private establishment, to which most of the inmates return if they have a relapse, its superintendent has the opportunity of taking note of the ultimate fate of his patients.

For my part, I consider it to be not only advisable, but imperatively necessary, that local and general treatment should be combined whenever the disease tends to repeated relapses, in spite of careful protection of the skin from noxious influences. It is of but little advantage to a patient to be discharged from a hospital cured, if his disease is going to be as bad as ever again in a few weeks. We need not search far to find innumerable examples, in which, in spite of the skill of practitioners familiar with the most approved and newest modes of treating diseases of the skin, persons have remained afflicted with eczema for years, now suffering from the eruption, now resorting to treatment, but seldom remaining free from it for any length of time. No general rules can be laid down for the constitutional treatment of those eczemas which depend upon a herpetic diathesis; but in special instances it is not very difficult to ascertain the suitable remedy, since the individuality of the patient, his physical habit, and his mode of life, furnish a clew to the means by which his constitution may be modified effectively and at the same time without injury. It would be unfortunate if the conviction, that the effect of *Hebra's* treatment is often merely palliative, were to lead physicians back again to the old routine of systematically purging all forms of this disease; but I do not hesitate to declare that I regard *Hebra's* assertion, that, "in chronic eczema, the mere use of purgatives never does good, and often does harm," as quite inaccurate. When chronic eczema attacks a corpulent patient who eats and drinks more than enough for the requirements of his system, and who is fond of fat food, and of food rich in fat-producing elements, he will recover more quickly, and the cure will last longer, if placed upon a methodical course of laxatives combined with suitable regulation of his food, than if his treatment be merely local and no attention be paid to diet. In such cases, where the eruption is very extensive, I often prescribe *Zittman's* decoction—in spite of its absurd composition—simply because patients follow out the directions for its use with such punctilious and almost superstitious exactness. But there is another class of habitual sufferers from eczema, whose idiosyncrasies are exactly the opposite of those described above, and who, without being really sick, are poorly nourished and much emaciated. To purge such patients as these, and to reduce their diet, would be to make the evil worse. On the contrary, they require a treatment calculated to augment the supply of nutriment, and to diminish the wear and tear of the system, such as the exhibition of cod-liver oil and similar medicines. These suggestions must suffice. It would take too long were we to attempt to detail all the indications for internal treatment which may accompany the topical applications, and which are deducible from the constitutional peculiarities of the patient.

CHAPTER VIII.

IMPETIGO—DIFFUSE SUPERFICIAL DERMATITIS, WITH FORMATION OF SMALL PUSTULES.

Etiology.—In impetigo, as in eczema, a serous exudation is secreted upon the surface of the corium, but at the same time young cells form in great abundance and are mingled with the serum. Hence, the contents of the vesicles which arise in impetiginous dermatitis are not limpid and transparent, like those of eczema, but turbid and yellowish, and, upon rupture of their epidermis, a purulent liquid runs out from them, which afterward dries up into yellowish or greenish crusts.

As the serum in the vesicles of eczema also contains a few young cells, and since in eczema impetiginosum the contents of some of the vesicles are yellow and puruloid, owing to the copious admixture of young cells, it is plain that no distinct boundary line can be drawn between eczema and impetigo; and that there are intermediate forms to which either name might be applied with equal propriety. This is especially the case in the moist eruption upon the scalp of children which sometimes bears the title of eczema capitis or faciei, and sometimes that of impetigo or porrigo faciei or capitis.

The first cause of impetigo which we shall mention is the direct action of irritants upon the skin. The more delicate the skin, so much the more easily do even slight irritants cause exudation, and proliferation of cells upon its surface. In some persons, the application of a pitch-plaster or of a poultice is sufficient to provoke an eruption of impetiginous pustules. This "vulnerability" of the skin is most common among people with a thin epidermis and clear complexion; but, above all, among scrofulous individuals. Among the latter, indeed, impetigo often appears without any appreciable irritation of the skin, and, together with chronic catarrh, and enlargement of the lymphatic glands, is one of the most decided symptoms of scrofula. Finally, impetigo attacks children without perceptible irritation of the skin, scrofulous diathesis, or other assignable cause. In such cases, which are not at all rare, it is generally assumed that the food is too nutritious, that the mother's milk is too rich, or that the eruption has been caused by the presence of an "acrid" material in the blood. No sound reasons can be advanced in support of such an idea.

Symptoms and Course.—The inflammation of the papilla, from which the exudation proceeds, is accompanied by a sense of itching, and by a desire to scratch. These subjective phenomena, and the objective ones, of small acuminated pustules (psydracia) upon a reddened base, and of yellow scabs, are about the only symptoms pre-

sented by impetigo. It is very rarely, and then only when the disease is very violent and acute, that impetiginous dermatitis is attended by fever and by general constitutional disturbance. According as the eruption is limited to a small part of the surface, or extends widely over a large area, it is called impetigo *figurata*, and impetigo *sparsa*. The former variety is most often seen upon the face, especially upon the cheeks, lips, nose, and scalp; but its appearance upon the trunk and extremities is also not uncommon. At first red specks, which may be either discrete or confluent, are observed upon rounded, oval, or irregularly-shaped spots of varying size. Extreme redness of the skin, with tension and glistening, accompanied by fever, constitutes a variety of impetigo, to which *Willan* has applied the name of impetigo *erysipelatodes*. Upon the reddened base, minute yellow points immediately develop, which soon grow to the size of small lentils, and rise a little above the level of the surrounding parts. After a day or two, perhaps sooner, the pustules burst and discharge their contents, which dry up into yellow crusts. Beneath these, the formation of pus continues, causing a gradual thickening of the crusts, while new pustules continue to spring up at their edges. At first, upon removing these crusts, which sometimes attain considerable thickness (impetigo *scabida*), we find beneath them the naked corium bathed in puruloid liquid; but, toward the end of the disease, a new, delicate covering of epidermis appears, through which the redness of the cutis is visible, and in which slight cracks form when the part is moved. The course of impetigo figurata usually is subacute, so that the process terminates in two or three weeks by detachment of the crusts. However, it sometimes lasts for several months, and even for several years, although this is not very common. In such instances, the substance of the corium takes part in the process, and becomes thickened and indurated just as it does in chronic eczema. The favorite seat of impetigo sparsa is upon the extremities; and it often covers an entire limb, sometimes, indeed, extending over the whole surface of the body. The reddening and itching of the skin, the appearance and bursting of the pustules, the formation and thickening of the crusts, and the continued development of new pustules around them, and, finally, the shedding of the crusts, all occur just as in the form previously described; only impetigo sparsa more frequently assumes a chronic character, and, since the cell-formation is not confined exclusively to the surface of the corium, but is also going on within it, it is more apt to result in superficial ulceration.

Treatment.—Many cases of impetigo require no treatment at all, and heal of themselves in a few weeks. Hence, in recent cases, we may confine our interference to softening the crusts with fresh butter

or other grease, when they grow too thick and hard, and to removing them with a poultice. But, if the disease already have lasted for weeks or months, expectant treatment is no longer appropriate, and we must consider whether to treat the disease locally or generally, according to the principles laid down in the previous chapter. The latter is more frequently indicated in impetigo than in eczema, as this form of dermatitis often constitutes one of the symptoms of a constitutional disease. In the treatment of impetigo, as a rule, the same local remedies may be employed which have already been proposed for the treatment of eczema. The disease, however, is not so tolerant of strongly irritating applications as eczema is, on account of its more inflammatory character, as is shown by the suppuration which characterizes it. White precipitate, the oxide and the sulphate of zinc, and slight cauterization, with nitrate of silver, are usually to be preferred to the preparations of sulphur, the soft soap, and tar.

CHAPTER IX.

ECTHYMA—DERMATITIS, WITH FORMATION OF LARGE, ISOLATED, PERMANENT PUSTULES.

Etiology.—Although the pustules of ecthyma always arise from a brightly-reddened, swollen, and infiltrated base, yet, in spite of the intensity of the inflammation, the suppuration is usually confined to the surface of the cutis, and is not apt to involve its parenchyma. In the latter case only does ecthyma cause ulceration of the skin, and produce scars, owing to contraction of the young connective-tissue cells by which the loss of substance is supplied.

Ecthyma often proceeds from direct irritation of the skin. The pustules caused by tartar-emetic ointment (ecthyma *antimoniale*), and those which appear upon the hands and arms of smiths and masons, from the contact of lime, or of detached particles of red-hot iron, are of this class, as well as the large pustules which arise from the violent scratching provoked by the presence of parasites, or by the itching of other eruptions. In other instances, ecthyma arises without any appreciable previous irritation during the course of febrile diseases, assuming a form somewhat analogous to what is generally called hydroa febrilis. Finally, ecthyma is also seen in persons broken down by privation, or other cause of exhaustion, or who have become cachectic by a physical drain upon the system, by severe or tedious illness, and by an abode in prisons, or in other unwholesome dwellings; as well as among drunkards and in scorbutic individuals. We shall treat of syphilitic ecthyma hereafter.

Symptoms and Course.—The inflammation and infiltration of the skin, which precedes and accompanies the development of ecthyma pustules, are usually attended by lancinating pain, and in irritable persons by fever. The pustules are scarcely ever very numerous. They are isolated, surrounded by a broad, red areola, and are most frequently situated upon the extremities, the seat, the bosom, the throat, and, far more rarely, upon the face. These pustules are hemispherical, project distinctly above the level of the surrounding skin, and are usually somewhat larger than a pea (*phlyzacium*). Their contents consist of a yellow, purulent liquid, often mingled with blood. After a few days, the contents of the pustules dry up, forming round, brownish scabs, which either remain flat, and soon fall off, or else, where the suppuration continues beneath them, they gradually grow thicker, and adhere for a longer time. In the first instance the scabs, after falling, leave behind them red spots, covered with new epidermis, and in the latter they leave ulcers, which, although usually shallow ones, sometimes eat deep into the skin. The course of ecthyma is sometimes acute, sometimes chronic. In the acute form, induced by external causes, or in that which arises symptomatically in febrile disease, there is generally but a single eruption of pustules. Each pustule lasts but for a short time, and the scab soon falls, leaving no excoriation, or at most a very slight one. In the chronic form, which is peculiar to cachectic ecthyma, successive eruptions of pustules take place at varying intervals; the redness of the surrounding areola of inflammation is often livid, and the contents of the pustules are bloody, or discolored (*ecthyma luridum*). Moreover, beneath the thick scab, which is long in forming, and late in falling, very obstinate ulcers develop, which often penetrate deeply into the cutis.

Treatment.—The only species of ecthyma which requires active treatment is the chronic variety, with tendency to ulceration of the skin. First of all, the cachectic condition must be corrected, if possible, by means of proper ventilation, generous food, the use of wine, good beer, and the preparations of iron and quinine. Externally, while the inflammation continues, warm poultices must be applied; afterward, when the ulcers are indolent, they require stimulation, especially by touching their surface with nitrate of silver.

CHAPTER X.

PEMPHIGUS—POMPHOLIX—SUPERFICIAL DERMATITIS, WITH THE FORMATION OF LARGE ISOLATED BLEBS.

Etiology.—In pemphigus, large blebs (bullæ), filled tensely with clear liquid, form upon a base which is slightly reddened but not infil-

trated. They resemble the blisters produced by vesicating plasters, or by chafing of the skin. We have no satisfactory explanation of the cause of this curious disease. In new-born children, pemphigus is nearly always, if not absolutely always, of syphilitic origin. Children are more frequently attacked than adults. Some persons exhibit signs of a general cachexia prior to the outbreak of the pemphigus; while others retain their blooming appearance, and feel perfectly well throughout the disease, unless exhausted by its repeated recurrence and by loss of sleep. Pemphigus sometimes assumes an epidemic form.

Symptoms and Course.—The first change noticed in the skin consists in the appearance, upon the back, belly, and extremities, of red circular spots, which itch and burn. In a few hours, at the middle of the spots, small transparent vesicles arise, which quickly enlarge and soon cover the whole of it, or else merely leave a narrow margin unoccupied. The vesicles are round or oval in shape, and of the size of a pea, a cherry, or perhaps of a walnut. At first their contents are transparent, afterward they become turbid and curdy. In two or three days the blebs burst, leaving an excoriation. For some days this continues to discharge, and then becomes covered by a scab, under which a new epidermis forms. A stain of pigment remains to mark the spot where the bulla was situated. Before the first blebs heal, new ones form; these are succeeded by others, and thus successive crops continue to spring up; so that we are often able to study all the stages of pemphigus simultaneously upon one patient. In some cases this only continues for a few weeks, and then ceases. But even then there is usually another attack, the whole process repeating itself after a lapse of weeks or months. These relapses, which are often three, four, or even more in number, resemble the first attack in their course and duration. In other instances, the formation of new blebs does not cease at the end of a few weeks, but continues for months, and even for years. Although at the beginning of the disease the general health of the patient is entirely undisturbed, yet as it progresses he grows pale, thin, and debilitated, either because the discharge exhausts him, or because the unknown cause of pemphigus exerts a noxious influence upon the entire economy. The emaciation, and pallid, cachectic look make all the more rapid progress, if rest at night be disturbed by the itching which attends the eruption of the blebs. Nearly all patients with chronic pemphigus finally die of marasmus.

The question has been actively debated, whether pemphigus be always chronic, or whether there be not also an acute pemphigus. This seems to me to be a mere dispute about words. When the erup-

tion does not last over two or three weeks, we certainly are warranted in calling it acute; but, if we choose, we may regard the interval when no blebs form, which often lasts for months, and during which the patient feels as well as before his first attack, as a period of "latence," and call the series of relapses chronic pemphigus.

Hebra and *Cazenave* describe a truly horrible malady called *pemphigus foliaceus.* In this affection a very few, or perhaps only a single bleb forms. They are not as tensely filled as those of other forms of pemphigus, but have a tendency to spread. In pemphigus foliaceus the liquid constantly pushes further and further beneath the epidermis, until finally the entire skin looks as if it were flayed, or else is covered with a brownish-yellow rind. It generally takes a year for the disease to arrive at this pitch of progress. Meantime portions of the skin heal, but only to be attacked anew. The malady always terminates in death.

Treatment.—We can only treat the symptoms of pemphigus, since, in spite of the meritorious labors of *Bamberger*, who has detected ammonia in the recently-passed urine of pemphigus patients, as well as in their blood and in the contents of the blebs, the cause of the malady, and the nature of the cachexia, or dyscrasia, giving rise to the eruption, are quite unknown. We can merely support the strength of the patient until the disease subsides spontaneously, and can endeavor to postpone his exhaustion and ultimate death as long as possible. As in all similar cases, whatever tends to increase the waste of the organism must be avoided, and every thing must be furnished in abundance which can replace what is consumed, or which can retard the consumption of tissue. With regard to external treatment, *Hebra* prohibits the use of baths and ointments as expressly as he does the internal administration of specifics. However, he recommends sprinkling of the moist spots with dry vegetable powder, especially lycopodium-seeds.

CHAPTER XI.

RUPIA—DERMATITIS, WITH FORMATION OF ISOLATED FLAT VESICLES, FROM WHICH SCABS OF A PECULIAR SHAPE ARE FORMED.

Etiology.—Rupia, like pemphigus, forms isolated blebs. Those of pemphigus, however, soon burst, while the rupia blebs last longer. Their contents become purulent, and often are bloody, and, after a while, dry up into a scab. Having once formed, the scab is gradually thickened by the addition of fresh exudation, which generally proceeds from ulceration of the deeper parts of the skin, and which also dries

into a crust, while upon its edges a border of bullæ forms, the contents of which dry up like those of the first one. Thus, the peripheral part of the scab is flatter than the older portion; the scabs of rupia are thick in the middle and flat at the edges, bearing some resemblance to oyster-shells. In most cases, but not in all, rupia is a symptom of constitutional syphilis. The causes of non-syphilitic rupia are unknown. Like ecthyma, it occurs most commonly in broken-down and cachectic subjects.

Symptoms and Course.—The usual seat of non-syphilitic rupia is upon the extremities. The blebs are isolated, and have a reddened base. They are not tightly filled, and their contents at first are limpid, afterward yellow or reddish. The scabs which form are of a dark color, and, after lasting some time, assume the shape described above. According as the thickness of the scabs is slight or considerable, they are distinguished into rupia *simplex* and rupia *prominens*. Removal of the scab exposes either an excoriation, or else a deep ulcer, over which a new scab soon forms. Sometimes the base of the bleb not only ulcerates, but becomes gangrenous (*rupia escharotica s. gangrenosa*). In such case the contents of the blebs are discolored or blackish. Beneath them, or beneath the scab, we find the corium destroyed and converted into a foul ulcer, covered with *débris* of the tissue, and indisposed to heal. Rupia simplex and prominens usually recover, leaving behind them a shallow scar, which is often stained with pigment; while rupia gangrenosa may cause death from exhaustion, or may accelerate the end of the already exhausted patient.

Treatment.—Our principal task in the treatment of rupia consists in combating the constitutional vice upon which it depends. If we succeed in this, young epidermis soon forms beneath the scabs, and the ulcers heal. But if we do not succeed in improving the constitution, local treatment will generally be found useless also. Besides the constitutional remedies, the scabs may be softened by poulticing. The ulcers which remain require stimulating applications, such as repeated touching with lunar caustic.

CHAPTER XII.

PSORIASIS—CHRONIC DERMATITIS WITH INFILTRATION OF THE CORIUM, AND ABNORMAL GROWTH OF EPIDERMIS.

Etiology.—In psoriasis, the effusion which forms upon the surface of the corium is insufficient to elevate the epidermis into vesicles. On the contrary, this form of dermatitis, which is always chronic, consists merely of a hyperæmia and infiltration of the skin, capable of

causing the papillæ to produce an unhealthy epidermis, which, mingled with the exudation, scales off in large flakes. The cause of psoriasis is quite unknown. This disease, which is of very frequent occurrence among all classes of society, cannot be regarded as the exponent of a dyscrasia, for it attacks perfectly healthy subjects, and, in fact, seems to prefer them, while invalids and decrepit persons often remain exempt. In some families psoriasis is hereditary. Men and women seem to be about equally liable to it, while it rarely attacks either children in their first year or very aged persons.

Symptoms and Course.—The disease always commences in small round spots upon the skin. These are reddened; project slightly above the surrounding level, and immediately after their appearance are smooth, looking as if the covering of epidermis had been raised by a serous effusion, and had sunk back again after the liquid had been reabsorbed. This small, round, red, infiltrated spot soon becomes covered with dry, white scales. According to *Hebra's* apt and simple description, it is from this form of psoriasis—psoriasis *guttata*—that all the other varieties of psoriasis arise, developing sometimes through extension of the morbid process, sometimes through retrocession of the disease at the point first attacked. An enlargement of the affected spot converts a psoriasis guttata into a *p. numularis;* this latter, when the process begins to decline in the central and older portion of the area of disease, so that the scales grow thinner and commence to fall off, becomes a *p. scutellata*, and this again becomes a *p. annulata*, when the redness disappears from the centre, and the skin at that point resumes its healthy appearance (*lepra vulgaris*, *Willan*). When the circles thus formed touch one another, interruptions occur at the points of contact, so that at last there merely remain segments of circles, producing a new form of the disease, called *p. gyrata*. Finally, *p. conferta s. diffusa* is the result of the confluence of numerous spots of the eruption. The favorite seat of psoriasis is upon the dorsal or extensor surfaces of the extremities, and, above all, upon the knees and elbows. The disease often remains confined to these regions, no trace of it being discoverable elsewhere upon the body. In cases of this kind, and indeed in cases where the eruption is quite extensive, it is often arranged upon each half of the body with remarkable symmetry. This, however, occurs in other cutaneous diseases also; for instance, in eczema. In very rare instances, psoriasis is confined to the eyelids, or to the lips, the prepuce, the scrotum, or to the labia majora: and there is no reason why these varieties should not be called *p. palpebrarum*, *labiarum*, *præputii*, *scroti*, or *pudendorum*, etc. But neither the diffuse nor the circumscribed form of psoriasis *palmaris* and *plantaris* is included in this series of varieties. In the diffuse form, the

palm of the hands and soles of the feet are reddened and covered with dry scales. We have ascertained that this is really a species of eczema. The circumscribed variety is always of syphilitic origin, and is to be spoken of hereafter. The circumstance that the morbid process from which psoriasis arises does not act continuously upon one spot for any great length of time, is not only the reason for the peculiar figuration of the eruption, but also accounts for the rarity with which degeneration of the deeper layers of the skin occurs in this disease. The exceptional cases in which the eruption does not soon subside, and in which the skin is thickened, rigid, and fissured, are called psoriasis *inveterata*. The diffuse or irregularly extended variety of the disease is the one most apt to assume this form.

Treatment.—Although it seldom is possible to effect a radical cure of a psoriasis, yet the treatment which merely aims at a temporary abatement of the eruption is very satisfactory. The objections which exist against the local treatment of certain forms of eczema and impetigo are not valid in case of the disease in question; psoriasis is never of constitutional origin, and never appears vicariously for other diseases. Moreover, vigorous local treatment is tolerated much better in psoriasis than in any other cutaneous affection hitherto described. *Psoriasis must always be treated locally and with the most energetic remedies.* Instead of the white-precipitate ointment, and the preparations of zinc and of lead, which often suffice to effect a cure of an eczema or an impetigo, and which, indeed, are often the only remedies applicable to the case, the articles best adapted to its treatment are green soap, tar, and the preparations of sulphur. It is best to commence the cure with one or two vapor-baths, in which, by means of soap and a moderately hard brush, the skin is to be cleansed as much as possible from the scales which adhere to it. If these vapor-baths cannot be had, the same object may be obtained by means of long-continued warm-water baths. After removal of the scales, the green soap is to be rubbed in twice daily, for a period of from three to six days, or else it must be applied upon a compress, which is to be renewed twice in the day. While this is going on, it is best that the patient should be enveloped in a woollen blanket, and that he should remain in bed, and in a well-warmed chamber. The treatment should then be suspended for about three days, during which the inunctions should cease, and the compresses may be laid aside. At the end of the interval, the patient must take another vapor-bath, or long-continued warm-water bath. If the infiltration of the cutis has not yet disappeared, the process is to be repeated. If, however, all the diseased spots seem soft and pliant, we may proceed to the application of tar or of tar-ointment (see page 476). We have already said that we prefer a solution

of tar and green soap (āā ℥ j), in alcohol (℥ ij), to the pure tar or the tar-ointment. If, from the first, there has only been a moderate degree of infiltration of the cutis, we may begin with two or three daily applications of the above solution immediately after the bathing and removal of the scales; and we shall nearly always effect our object in two or three weeks by this means. Similar results, of which, however, I have no personal experience, may be obtained by the use of ointment of iodide of mercury or of sulphur and iodide of sulphur. For a time, *Hebra* used to recommend the application of a concentrated solution of sulphuret of lime, in psoriasis (*Vleminckx's* solution). (℞. sulphur. ʊtrini ℔ ij, calcis vivæ ℔ j, to be boiled in three gallons of water down to a gallon and a half; the liquid to be filtered when cool.) *Hebra* orders the solution to be rubbed vigorously into each diseased spot, upon a bit of flannel, until all the scales have come off and the papillæ are exposed. He then puts the patient in a warm bath, where he is to remain for an hour. After the bath, the diseased spots are to be rubbed with any unctuous substance (such as cod-liver oil) or else with tar-ointment or ointment of chrysophanic acid.

The above plan of treatment is adapted for psoriasis in its more severe and extensive forms. Pencilling the affected spot with a solution of corrosive sublimate (hyd. chlor. corrosiv. 3 j; alcohol ℥ j) is an excellent way of treating slighter efflorescences of the disease. Such pencillings have the advantage of being much simpler, but they cause such severe pain that they cannot well be used where the eruption is at all extensive.

Although the quickest and surest way of treating psoriasis is by making topical applications, yet it cannot be denied that the same object will be attained by the internal exhibition of arsenic. In view of this, as well as of the fact that the treatment just described is quite unable to prevent relapses, and bearing in mind the well-known harmlessness of treatment by arsenic, if carefully conducted, I hold it to be doctrinary and incompatible with the interests of the patient to adopt one or other method of cure exclusively, and not to combine the two. Arsenic is usually given in the form of *Fowler's* solution. At first, six drops are to be taken daily; and the dose is to be increased one drop every fifth day, until it amounts to about thirty drops. *Veiel* uses the "Asiatic pills" almost exclusively, because he can regulate the dose much better when the arsenic is in this form than when it is in the form of drops. He dissolves white arsenic in boiling water, mixes it with bread and pepper, and makes it into pills, each of which contains the thirtieth of a grain of arsenic. Of these, at first, he gives three daily, gradually raising the dose to eight or nine pills daily (that is, to a quarter of a grain of arsenic per diem). As soon as any sense of

oppression occurs in the region of the stomach, or when tears begin to flow from the eyes, the treatment is to be suspended for a few days. Cantharides, the antimonials, anthracokali, tar-pills, and other articles, formerly in vogue, having proved themselves worthless in treatment of psoriasis, have very justly been abandoned of late years. However, in plethoric, vigorous subjects, the cure may be accelerated by restricting the diet, and by combining the use of laxative tisanes with the specific treatment; for we know that such measures alone, when pushed vigorously, will cause a psoriasis to disappear.

CHAPTER XIII.

LICHEN—DERMATITIS FORMING GROUPS OF PERSISTENT CONICAL NODULES.

Etiology.—In lichen, instead of the appearance of vesicles, filled with serum, upon the skin, there is an eruption of solid nodules (papulæ). The exudation which forms in this disease infiltrates the cutis, and produces a circumscribed swelling of its parenchyma, but the portion of it effused upon the free surface of the cutis is only sufficient to swell the cells of the rete Malpighii, and to loosen the layer of epidermis above it. This is why lichen terminates by desquamation. It is only in a few cases that the source of lichen can be traced to the action of slight external irritants; for instance, to the bites of parasites, to chafing of the skin by roughness of the clothing, to the effect of dirt, or to the influence of high temperature. In most cases we cannot tell why the numerous small circumscribed portions of the cutis, of which the lichen-knots consist, should be thus affected, while the rest of the skin about them appears sound. Lichen is especially common among scrofulous persons.

Symptoms and Course.—Lichen is characterized by nodules of about the size of a millet-seed. They are of normal color, or else of a pale, yellowish red; sometimes they are even paler than the surrounding skin. In the latter case, the blood-vessels, from which the exudation originally proceeded, have afterward been compressed by the exudation. The nodules usually form groups; sometimes they only appear upon particular regions of the body; at others, they spread over a wider extent of surface. In the milder forms of lichen (*lichen simplex*), the papules itch but slightly, or give no annoyance whatever, and only last for a short time. They disappear in a week or two, and, after desquamation of the epidermis covering the papules, the disease usually terminates. More rarely, lichen simplex assumes a chronic form, fresh eruptions of the nodules following one another in rapid

succession. In the severer form (*lichen agrius*), the outbreak is sometimes accompanied by fever, and by general disturbance. The nodules are often crowded closely together, and stand upon a reddened base. They are likewise red themselves, and itch and burn intensely. The inflammation readily becomes aggravated, so that in a few days the lichen changes into an eczema rubrum. The course of this variety may also be acute, and terminate in recovery at the end of a week or fortnight; more usually, however, it becomes chronic.

Hebra—who never applies the term lichen to a papular eruption unless the papules retain their solid form from beginning to end of their course, and do not transform into vesicles or pustules—describes lichen *ruber* as a disease which, in its later stages, bears some resemblance to psoriasis. Here there are red nodules of the size of a millet-seed, which are covered with thin scales, and which at first are separate, and do not itch nor extend at the periphery. With every succeeding outbreak of the eruption, the part becomes more thickly studded with nodules, and the intervening surface grows smaller, until at last the edges of the papules touch one another, and form large, infiltrated, red patches. Finally, the entire cutaneous surface may become the seat of such a papular eruption, and of the diffuse infiltration arising from it. Like all other universal exanthemata, this has a prejudicial effect upon the general health of the patients, the majority of whom die in a state of marasmus.

Treatment.—The slighter forms of acute lichen require no treatment. In especially obstinate cases we may have recourse to the exhibition of arsenic. *Veiel* believes this to be of most service in all those cutaneous diseases in which the cutis is infiltrated, while in other forms it is much less efficient. Cold applications and the exhibition of laxatives are the suitable remedies for lichen agrius; but blood-letting, which likewise has been recommended, should never be practised. Chronic lichen agrius often defies the most active treatment. Baths, soft soap, tar, and the preparations of sulphur, but especially the internal administration of arsenic, are the remedies most to be advised.

CHAPTER XIV.

PRURIGO—DERMATITIS WITH SMALL, SCATTERED, ITCHING NODULES.

Etiology.—The flat nodules which are peculiar to prurigo have the color of the surrounding skin. If punctured, they usually discharge a drop of clear liquid, while the puncture of a papule of lichen is followed by the flow of a drop of blood. Upon squeezing a prurigo nodule laterally, we observe that the epidermis becomes stretched

and transparent, and that a watery liquid appears. From these peculiarities of the nodule, and from the results of the treatment of prurigo, *Hebra* concludes that in this disease we have to deal with a liquid effusion in the deeper layers of the epidermis, which, however, is not profuse enough to form vesicles, but merely produces flat nodules, at first perceptible to the touch, and afterward to the eye. The subjective symptoms and course of the disease, especially the intolerable itching which accompanies it, seem fully to substantiate the correctness of this hypothesis, which, indeed, cannot be demonstrated directly upon the dissecting-table, as the lesions described above always disappear after death. In spite of the identity of their symptoms, it is usual to discriminate between the form of prurigo provoked by lice, ticks, irritating dust, and other external influences, and that very obstinate disease known as true prurigo, which, arising without known cause, often continues throughout life. Want of proper care of the skin and scanty food seem to play an important *rôle* in the etiology of true prurigo, which is much more common among the poorer classes than among the well-to-do. The disease appears at every age, excepting only the first years of childhood. Men are more liable to it than women.

Symptoms and Course.—The most prominent objective signs of prurigo consist in those resulting from the violent scratching, and not in the nodules themselves, which are scattered and flat, and, indeed, are often hard to find, although, from the irritation which they produce upon the skin, they create an itching as intolerable as that produced by the bites of insects, or by gently tickling the surface of the skin with the tips of the fingers, or with a foreign body. Scratching with the nails tears off the epidermis from the nodules; a slight bleeding is the result, and the blood effused dries into a crust. It is these innumerable little crusts which remain after the papule has disappeared that form the most conspicuous object upon the skin of a patient with prurigo. As scratches and papules are also induced by the bites of vermin, we must always ascertain in such cases whether we have to deal with lice and itch-insects, or with genuine prurigo in its stricter sense. Gross errors, especially the mistaking of the itch for prurigo, in persons whom one would not be apt to suspect of having the itch, are of daily occurrence. One of the most important distinctive points consists in the locality upon which the papules and scratches lie thickest. In prurigo the principal seat of the papules and scratched places is upon the extensor side of the limbs, especially upon the legs—which is never the case in the itch—and they are quite as numerous upon the back as upon the belly. Body lice, on the other hand, lodge chiefly where the shirt is thrown into folds, as about the neck and

waist-band, while the favorite harbor of the itch-insect is upon the flexor sides of the limbs, between the fingers, and upon the belly, where the prurigo-papules do not often come. However, the positive discovery of the tracks of the itch-insect, or the detection of the insect itself, or the presence of lice, or their nits, furnishes a criterion for distinguishing between scabies, prurigo pedicularis, and true prurigo, better even than that afforded by the seat of the papules and excoriations. But even in the face of such testimony as this, when the itching is of very long standing, and involves an unusually large extent of surface, we should bear in mind the possibility of a complication of these diseases, the more so since persons of the class most liable to prurigo are equally likely to be infested by lice and the itch-insect. The dark stain, seen upon the skin of patients who have long suffered from prurigo, is a consequence of the scratching, and is of no service as a diagnostic mark, since it is nearly always seen upon the skin of people who have long been infested by vermin. Slighter grades of prurigo are called prurigo *mitis;* the severer grades are called prurigo *formicans*, from the resemblance of the sensations to the intolerable itching and burning produced by the contact of a multitude of ants. Prurigo *ani*, which is limited to the region of the anus, and that form which appears upon the labia majora in women, and upon the penis and scrotum in men—or prurigo *pudendorum*—is intermediate between prurigo and eczema. As has already been stated, this is a most obstinate affection, and may last with undiminished intensity for months, and even for years. Generally, however, it grows worse during the autumn and winter, and makes a remission during the spring and summer. The itching is usually most distressing in the evening, and at night. So constant are the disquiet and torment of the patients, who are often deprived of their sleep night after night, that, driven to desperation, they sometimes commit suicide, while others become insane. On the other hand, the general state of the nutrition does not often suffer; at all events, not until very late in the disease. Any premature marasmus which may exist is much more frequently dependent upon complications, or upon the needy circumstances of the patient (cachexia pauperum), than upon the prurigo itself.

Treatment.—The radical cure of prurigo is a task so difficult that it is rarely accomplished. On the other hand, it is nearly always possible to produce a temporary abatement of the symptoms, and sometimes, indeed, to effect a complete although transient relief, by stimulating the process of desquamation of the cuticle by means of cutaneous irritants, thus accelerating the process of its regeneration. Hence, baths or lotions containing a solution of common salt, potash, corrosive sublimate, lime-water, or dilute acid, inunctions of tar-oint-

ment and of soft soap, and vapor baths, are preferable to the tepid baths of bran-water, milk, or mucilage. A very sure remedy for prurigo—although merely a palliative one—consists in friction of the skin for half an hour at a time with a piece of flannel dipped in the solution of sulphuret of lime mentioned above (see p. 488), and known as *Vleminckx's* solution. After being rubbed down, the patient is to be put in a bath, in which he must spend at least an hour. It is a good plan to follow the bath by a shower-bath, and then to rub the body with oil. Even the first bath always affords great relief, and the itching generally subsides completely after continuing the bathing for a week. Unfortunately there is always a relapse, rendering a repetition of the treatment necessary. Any complication which may exist must be combated by internal medication and by suitable diet; and, when the nutritive state of the body is deranged, that also must be regulated, if possible. *Veiel* regards arsenic as a real specific in prurigo, and claims never to have used it without effect.

CHAPTER XV.

ACNE, ACNE VULGARIS, ACNE DISSEMINATA—INFLAMMATION AND SUPPURATION OF OBSTRUCTED SEBACEOUS FOLLICLES.

Etiology.—*Bärensprung* very properly classes inflammation and suppuration of the sebaceous follicles with similar derangement of the mucous follicles; and in truth the acne pustule of the skin and the follicular ulcer of the mucous membrane both originate by the same process—obstruction of the outlet of a follicle, inflammation of its walls, and bursting of its covering externally. By and by we shall consider the effects of obstruction of the follicles by inspissated sebaceous matter upon which the development of the so-called *comedones* (Mitesser) depends. A comedo is not an acne at first, and does not become one until the obstructed follicle inflames. In most cases such inflammation terminates in suppuration, producing *acne pustules.* But it sometimes happens that the inflammation is resolved, and then only leads to chronic infiltration and thickening of the wall of the follicle, or, in other words, to the formation of *acne nodules* or *tubercles.* Very few persons ever remain perfectly exempt from acne vulgaris. The majority, however, merely suffer from it during the period of puberty, and the form of the disease is mild, and, even in individuals who have been afflicted by acne vulgaris in its worst form for years, the affection has never commenced in childhood, but only appears at the time of adolescence, although it may considerably outlast that period. It is, however, extremely rare for acne to last until the age of advanced man-

hood. Although the male sex is more predisposed to acne than the female, yet, to the dismay of vain mothers, "a bad complexion"—the usual euphemism for acne—often disfigures their daughters, at the very time when they are about to be introduced into society. Hence, it can hardly be denied that some connection does really exist between the development of acne and the process which is going on in the sexual system. The laity go further; and, according as they are inclined to regard mankind from a gloomy or a favorable point of view, they ascribe acne to masturbation or to sexual excess, or else attribute it to excessive chastity and over-continence.

Symptoms and Course.—The most frequent seat of acne is upon the face; next in frequence it appears upon the back, bosom, buttocks, and upper arms. The disease commences by the reddening and swelling of the skin at a circumscribed spot, in the middle of which there is a black point—the comedo—(*acne punctata*). If resolution of the inflammation takes place, the swelling subsides in the nodule, the epidermis, loosened by the inflammation, scales off, leaving a reddened and somewhat hardened spot which remains upon the skin for some time. After a while, however, this likewise disappears. If the inflammation passes on into suppuration, a small pustule forms at the apex of the nodule, and after a while bursts, leaving a yellow scab. In rare instances, the nodule upon which the pustule is situated disappears soon after the pustule forms, or else after it bursts; generally, however, it subsides slowly and gradually. It not unfrequently happens that the cutis surrounding the inflamed follicle is more extensively involved in the inflammation, and, becoming infiltrated, swells, hardens, and acquires a deep-red hue. Inflammation of the skin arising from this cause closely resembles furunculous dermatitis. This may end in resolution, the rounded, flat, broad, elevation of the skin into which the acne nodule has converted itself gradually subsiding, casting off its cuticle, and slowly losing its color; but it frequently terminates in suppuration. The follicle which in such cases has become loosened from its attachments is discharged just as the dead "core" of a boil is discharged, and a scar remains.

Treatment.—Acne is quite independent of "impurity of the blood;" and the "decoctions of woods" and the laxatives—even if they had any real title to their supposed properties as "blood purifiers"—would not be suitable remedies in this affection. But it is not enough that the physician should merely refrain from prescribing these articles; he must also warn his patients—who usually take them behind his back—of the impropriety of so doing. Other internal medication should also be abstained from, as it is useless, and since the treatment by external application is always effective. Great benefit is often obtained

by washing the acne nodules in solutions of caustic potash or of corrosive sublimate, or with tincture of benzoin. *Veiel's* treatment also, which is exceedingly efficacious, consists in vigorously brushing the nodules with soft soap and a tooth-brush. The preparations of sulphur are still more preferable; they are very generally used, and *Kummerfeld's* lotion enjoys a great reputation among the laity. (℞. sulphur. precip. ʒ ij, camphor. gr. x, gum. mimosæ ʒ j, aquæ calcis, aquæ rosar. āā ℥ ij.) The directions for the use of *Kummerfeld's* lotion are: after shaking it well, to apply it at bed-time to all that portion of skin affected by acne, and the next morning to rub off all the sulphur which adheres to the skin, without wetting it. *Hebra* directs precipitated sulphur to be mixed with equal parts of alcohol, carbonate of soda, laurel-water, and glycerine, so as to form a paste, and this is to be rubbed into the nodules which are previously to be energetically scrubbed with soap and water. This treatment is also to be practised in the evening, and then the paste may remain upon the tubercles through the night. In persons who can afford the time, the paste may be applied night and morning, and left continuously in contact with the acne nodules. Where matter has formed, it is to be evacuated by cautious punctures. If the inflammation be very intense, the best application is a warm poultice.

CHAPTER XVI.

MENTAGRA — SYCOSIS — INFLAMMATION AND SUPPURATION OF THE SEBACEOUS GLANDS AND HAIR-FOLLICLES OF THE BEARD.

Etiology.—Sycosis is an inflammation of the sebaceous glands and of the deeply-rooted hair-follicles of the beard, but the tissues of the cutis about these glands and follicles likewise take part in the inflammation and become the seat of intense hyperæmia, swelling, infiltration, and suppuration. The disease scarcely ever occurs in women and children, who never shave; but only affects adult males, particularly at a time when the beard has acquired a tolerably thick growth. Dull razors, awkward shaving, the effect of irritating soap, of snuff, or of dirt, seem to cause the disease, or, at all events, to predispose toward it. In the majority of cases, it has no assignable cause. I can confirm the statement often made, that mentagra sometimes proceeds from the penetration of the parasitical growth of herpes tondens and herpes circinatus into the large and deeply-seated follicles of the beard. I have seen a case in which a man, who had contracted herpes tondens from a sick cow, had mentagra a few weeks afterward. What was very characteristic in this case was, that the eruption, half of which

was in the beard, the other half without it, presented the features of herpes circinatus in the latter situation and that of mentagra in the former.

Symptoms and Course.—The disease commences with a sense of heat and tension, accompanied by the development of red infiltrated nodules, of the size of a pea or lentil, between the hairs of the beard upon the lips, chin, cheeks, and, in rare instances, between the hairs of the eyebrows and armpits. After a while, pustules, which are always perforated by a hair, appear upon the tips of the nodules. The pustules burst in a day or two, and pour out their contents, which immediately dry into brownish crusts. The nodules still continue beneath the crusts, and, even after the latter have fallen, they diminish very slowly or not at all. Although at first the eruption is scanty and scattered, yet, under the influence of successive attacks, the tubercles multiply gradually, until, from the close crowding of the nodules, together with the infiltration of the intervening skin, the appearance of the surface becomes suggestive of a fig—whence its name, "*sycosis.*" Sometimes sycosis is confined to single spots of no great magnitude; in other cases, every part of the face where the hair grows is covered with dusky-red nodules, pustules, and scabs. The disease is of indefinite duration, and often lasts for years and even tens of years without subsiding, but also without leading to other lesions. When it heals, the hair-follicles having been destroyed, the hair does not grow again upon the affected spot, which presents the appearance of a scar.

Treatment.—I used formerly to regard sycosis as one of the most intractable of diseases, as it generally resisted all treatment, notwithstanding the numerous remedies which have been proposed for its cure. In the last few years, however, during which I have adhered strictly to the treatment proposed by *Hebra*, in *Virchow's* hand-book, I have altered my opinion, and recommend the following procedure. In the first place, all the scabs are to be softened and removed by rubbing them with oil, or by the application to them of a rag saturated with oil. We must then insist upon the patient's shaving daily, in spite both of his opposition and his barber's. The pustules which appear after the shaving are all to be opened with a delicate scalpel; and where they are confluent, long incisions must be made through the clusters. Like the shaving, this scarification is far less painful than might be supposed, and it likewise must be repeated daily. In extraordinarily obstinate cases the opened pustules must be touched with concentrated acetic acid, or with a solution of corrosive sublimate, consisting of one part of the sublimate to two of alcohol. During the night it is generally sufficient to cover the affected part with a rag,

thickly smeared with white-precipitate ointment, or with a paste made of equal parts of sulphur, glycerine, and alcohol.

CHAPTER XVII.

ACNE ROSACEA—GUTTA ROSACEA—CHRONIC INFLAMMATION OF THE SEBACEOUS GLANDS OF THE FACE, WITH DILATATION OF THE BLOOD-VESSELS AND GROWTH OF THE CONNECTIVE TISSUE ABOUT THEM.

Etiology.—In acne rosacea the sebaceous glands of the face, especially those of the nose, become the seat of a chronic inflammation. The tendency to suppuration in this affection is much less than in acne vulgaris and sycosis; but, on the other hand, it is always accompanied by an inflammatory hypertrophy of the surrounding connective tissue, as well as by considerable dilatation of the blood-vessels about the diseased glands. Acne rosacea is particularly common among tipplers, especially among wine and brandy drinkers; although it is by no means so exclusively confined to such individuals as to warrant the inference that a person is intemperate because he has acne rosacea on his face. Women at the climacteric epoch, even though they never touch wine, beer, or spirituous liquor, are often afflicted by this disorder, which is all the more distressing to them because of the prevailing prejudices regarding its cause. Young women, also, with deranged menses, sometimes are attacked by acne rosacea.

Symptoms and Course.—The disease commences by the appearance upon the face of discrete coppery-red spots, whose color depends upon the varicose dilatation of small blood-vessels. Acne nodules then develop upon these bluish-red spots; but, even when pustules mature, the nodules do not decrease in size, but continue gradually to grow larger. The effect of this constant formation of new tubercles, and of the excessive hypertrophy of the adjacent connective tissue of the skin, is to give rise to most unsightly deformity. The nose, which is the usual seat of acne rosacea, becomes enlarged and misshaped, and in bad cases it is studded with knobs and humps of various size, and assumes a purple color. The forehead and cheeks are often similarly affected, and sometimes the disease involves the whole face.

Treatment.—As soon as acne rosacea begins to appear, the use of spirituous liquors is to be strictly prohibited. If there be any derangement of menstruation, it is to be treated according to directions already given. It is also advisable to endeavor to compress the vessels of the reddened spots by painting them with collodion. When this treatment is not well borne, *Veiel* obtains benefit from concentrated lead-water, with the addition of terra sigellata and a little cam

phor. When the acne nodules appear, the preparations of sulphur may be employed; but, instead of the paste recommended for the cure of acne vulgaris and sycosis, it is better to use *Kummerfeld's* lotion, or a turbid mixture of sulphur and water, or alcohol. In old cases, almost every treatment will prove ineffectual.

V.—*HÆMORRHAGES OF THE SKIN—PURPURA.*

Etiology.—All hæmorrhages of the skin proceed from a solution of continuity of the walls of the cutaneous vessels; blood never transudes through the wall of a blood-vessel, unless it be ruptured. Purpura are reddish, bluish, or blackish spots, the result of extravasation of blood into the parenchyma of the cutis, the blood filling up the interstices between the elements of the cutaneous tissues, but causing no swelling of the skin. When the purpura are small and round, they are generally called *petechiæ;* when elongated into stripes, they receive the name of *vibices;* and when diffuse and of irregular shape, they are called *ecchymomata.* If the effusion of a somewhat large quantity of blood causes the skin to swell in the form of small nodules, we have the *lichen lividus* (*Willan*), or the *purpura papulosa* (*Hebra*). The broader flat tumefaction of the skin caused by a more diffuse extravasation, and which resembles the wheals of nettle-rash, is called *purpura urticans.* Sometimes a hæmorrhage separates the epidermis from the papillary layer, forming blebs, *purpura bullosa;* in other cases the blood perforates the epidermis and appears upon its exterior. In the so-called "bloody sweat," blood really exudes through the pores of the skin. It is not mingled with sweat, however; and since, moreover, this bleeding has nothing to do with the secretion of sweat, the above term is inappropriate.

The causes of hæmorrhage into the skin are:

1. External injury. The traumatic hæmorrhages caused by the bites of fleas deserve some attention, since inexperienced physicians are occasionally misled by them, and are induced to diagnosticate some serious disease of the blood. Immediately after the bite of a flea, a roseola spot or wheal makes its appearance, in the middle of which the small, dark-red wound—*the stigma*—is perceptible.

Besides this, however, in persons whose blood is deficient in fibrin, a hæmorrhage takes place into the cutaneous substance, about the puncture, similar to that much more considerable extravasation which occurs around a leech-bite in persons of a similar constitution. These little hæmorrhages from flea-bites continue visible after the puncture has disappeared; so that, although the stigma may afford a distinctive

mark between a roseola or urticaria pulicosa, and other forms of roseola and urticaria, it cannot be used to distinguish between purpura pulicosa and other forms of purpura. Besides the discovery of fleas and of fresh flea-bites, the mere consideration of the region upon which the purpuric spots are situated will often enable us to estimate their origin correctly. Fleas generally lodge in positions from which they can crawl between the folds of the shirt. Hence, when we find petechiæ upon the neck and shoulders, and about the waist in women, while few or none are visible upon other regions, especially upon surfaces which are usually exposed, we may conclude that the hæmorrhagic spots are the vestiges of old flea-bites.

2. Hæmorrhage into the skin may proceed from over-distention and rupture of its blood-vessels. Thus, after severe coughing, or after violent vomiting, we often see the face speckled with purpuric spots; and, when the venous circulation of the lower extremities is obstructed, we often find purpura upon them, accompanied by varicosities of the veins. Even intense fluxionary hyperæmia seems sometimes to cause rupture of blood-vessels in the skin. At all events, the English physicians describe an affection called purpura simplex, which appears in healthy and robust subjects, particularly in those who indulge freely in spirituous liquors.

3. The most frequent source of hæmorrhage into the skin is a nutritive derangement of the walls of the blood-vessels. Under this head comes the *purpura senilis*, one of the symptoms of senile marasmus, as well as the purpura which appear in severe constitutional disease, such as typhus, small-pox, measles, and scurvy, and that which is the chief symptom of the *morbus maculosus* of *Werlhoff*. The *peliosis rheumatica*, a form of purpura accompanied by rheumatic pains, and which is only met with upon the lower extremities, is probably of this class likewise.

Symptoms and Course.—Red spots resulting from hæmorrhage may be distinguished from similar spots caused by mere vascular distention, by the fact that the latter disappear under pressure of the finger, while the former remain unchanged. They vary considerably in size, situation, and duration; but these variations, as well as the subjective symptoms which accompany them, depend principally upon the character of the original disease, or its complications. Hence, we must refer to the chapters upon typhus, small-pox, measles, scurvy, and morbus maculosus Werlhofii for a further account of these hæmorrhages, and shall now merely add a few words about peliosis rheumatica. This affection, first observed and recognized as a distinct disease by *Schönlein*, usually attacks young subjects with delicate skin, who have already suffered from rheumatism. It is generally accompanied by

fever. The patient first becomes aware of a pain in the lower extremities, particularly in the legs. Upon examination, the painful part is found slightly swollen, œdematous, and studded with red spots of the size of a millet-seed or lentil. At first the spots are bright red, and disappear under pressure of the finger, which shows that at this period they depend upon hyperæmia. They afterward assume a dirty-brown color, and then no longer are effaced by the finger, proving that blood has escaped from its vessels into the substance of the skin. This eruption of spots generally recurs by fits several times in succession, so that the disease usually lasts for several weeks, although, if the patient maintain a recumbent attitude, the first crop of spots usually fades out again in from four to eight days. In some cases the disease drags on for months, with repeated relapses.

Treatment.—In treatment of hæmorrhage into the skin, special attention must be paid to whatsoever constitutional disorder may exist. Besides this, it is customary in these cases, as in almost all other hæmorrhages, to give the mineral and vegetable acids internally, and to bathe the purpuric spots with dilute acids—especially with vinegar and water. The efficacy of these prescriptions is very problematical. In the treatment of peliosis rheumatica it is an important rule that the patient should be kept in bed, and in an uninterruptedly horizontal attitude, until the eruption disappears, and for some little time afterward.

VI.—*GROWTHS IN THE SKIN.*

We shall leave the description of a majority of the cutaneous growths to text-books of surgery, especially the account of carcinoma, sarcoma, and epithelioma, and shall confine ourselves here to a brief sketch of the disease known as lupus, a neoplastic formation peculiar to the skin, and to the mucous membrane of the nose, mouth, and fauces.

CHAPTER XVIII.

LUPUS—LUPUS EXEDENS.

Etiology.—The neoplasm, upon whose development in the skin lupus depends, is classed among the "granulation growths" (Granulationsgeschwulste), by *Virchow*. It arises in the form of nodules, which either remain distinct (*lupus tuberosus*), or else coalesce, causing a diffuse thickening of the skin (*lupus hypertrophicus*). The elements which form a nodule of lupus consist of small cells which greatly

resemble the cells of the rete Malpighii. The epidermoid elements also suffer "accidentally." The epidermis over the diseased region is very thin, and is usually covered with fine scales. Small white granules, like millet-seeds, are produced from the sebaceous glands; the hairs become deformed and humped from irregular deposit of the horn-cells, and perish as the disease advances. The neoplasm penetrates deeper and deeper below the surface; it advances from the skin to the subcutaneous tissues, and thence often attacks the cartilages and bones. As the malady goes on, ulceration generally sets in. Crusts form, under which the tissues break down into "devouring ulcers" (*lupus exedens*). But it sometimes happens that the lupus-nodules subside, their cellular elements undergoing fatty degeneration, followed by absorption. The site of the disease then becomes depressed, and is marked by a deep scar, although it has never been in a state of ulceration (*lupus non exedens*).

The etiology of lupus is quite unknown. It is true that the disease often attacks scrofulous persons; but there are many well-marked cases of scrofula who remain free from lupus, while quite as many, who never have been scrofulous, and who previously had seemed quite sound in other respects, are attacked by it. Its relations with congenital syphilis are very similar. It cannot be denied that lupus is tolerably common among persons whom we suspect, or whom we know, to suffer from hereditary syphilis; but, on the other hand, it is well established that the majority of persons so affected never suffer from lupus. Hence we are not warranted in distinguishing lupus into lupus scrofulosus, syphiliticus, idiopathicus, etc. Statistics as to the frequence of lupus at different ages, and in the two sexes, and in the various ranks of society, show that the malady is rare before the tenth year of age, and still more rare after the fortieth, and most common between the ages of ten and twenty; that both sexes are afflicted by it with about equal frequence, although perhaps women suffer a little more often than men; and, finally, that lupus is less frequently seen among the rich than among the poorer classes.

Symptoms and Course.—The most frequent seat of lupus is upon the face, especially upon the nose. Nevertheless, the disease may attack other regions, such as the neck, shoulders, chest, and extremities, especially over the joints. The first symptoms are often overlooked, or at least too little importance is attached to them. They consist in the formation of small, painless, brownish-red specks or nodules (*l. maculosus, s. tuberculosus*), which, although quite hard, are so remarkably vulnerable and tender, that they bleed readily; and with very slight force the point of a pencil of nitrate of silver may be made to penetrate them deeply. The disease may remain at this stage, with

out advancing for a long time, even for several years. It is only in rare instances that lupus takes on a subacute course, so as to produce extensive destruction in a few weeks. As the malady advances, its appearance changes. In some instances the nodules multiply and grow larger; their surface becomes tense and shining, and is covered with detached scales of epidermis (*lupus exfoliativus*). As the disease goes on, the hardness of the spots and nodules is resolved, and the affected portion of the skin, which formerly rose above the surrounding level, or which at least was even with it, now sinks gradually, and attaches itself firmly to the part beneath it; and, as the recovery progresses, shrinks and is converted into a white, hard, shining cicatrix. When the course of a lupus is as above described, large portions of the skin becoming transformed into a tissue resembling that of a scar, without any ulcerations having occurred, it is called *lupus non exedens*. Apart from the unsightliness of the scars themselves, the face may suffer great deformity, in this form of lupus, from contraction or dilatation of the nostrils, and from ectropion caused by retraction of the cicatrices. In other instances the outset of the disease is quite similar to the above; brownish-red spots and nodules form, which continually shed scales of dry epithelium. This condition, however, having continued for a variable period of time, the nodules begin to grow, the maculæ become papules; new nodules form, and while the surrounding skin becomes more hyperæmic, red, and shining; a superficial erosion occurs upon the summit of one of the nodules, the product from which rapidly dries into a scab. These scabs gradually grow thicker and broader by accretion of fresh matter at their base. If forcibly detached, a slight bleeding ensues, and we can perceive that there is a loss of substance of the skin, in which the middle of the scab is embedded more or less profoundly. This is the characteristic course of that form of the malady known as *lupus exedens* or *exulcerans*, and also as *l. rodens*, as well as *herpes rodens s. exedens*, *s. esthiomenos*. Sometimes the disease begins from a pustule, without the previous development of a reddish-brown spot or nodule. In such instances, lupus at its outset might easily be mistaken for a simple impetigo, and the error would only become apparent upon the discovery that there is a loss of substance in the cutis, beneath the scab. According to *Biett*, it is convenient to distinguish two forms of ulcerative lupus, a superficially destructive and a profoundly destructive form. The former often involves a large extent of surface, and occurs not only upon the face but upon the shoulders, and other parts of the body enumerated above. Sometimes the process subsides at the point first attacked, leaving a scar, which, by its ribbed and reticulate aspect, closely resembles the scar of a burn; meanwhile it continues to extend at the edges of the

cicatrix; the disease is then called lupus *serpiginosus*. The more deeply destructive form of lupus exedens begins almost exclusively upon the alæ nasi and the tip of the nose. It is accompanied by decided swelling and hyperæmia of the anterior part of the nose. The scab which forms upon these deeply-seated nodules is of great thickness, proportionate to the depth to which the destructive process has penetrated. Beneath this scab, which is continually renewed, the skin, subcutaneous tissue, cartilage, and even the bones of the nose, are destroyed, sometimes in the space of a few weeks' time, but more frequently in course of several months or years. Sometimes the disease, instead of commencing in the integument of the nose, first attacks the mucous membrane, causing great devastation of the interior of the nasal cavity, and even destroying the septum narium before the ulceration involves the skin.

It now remains for us to describe lupus hypertrophicus. The most frequent seat of this form of lupus is also upon the face, although it not unfrequently appears upon other parts of the body. I have known it to attack the face and extremities simultaneously. Here, too, numerous nodules of variable size are first observed; they are covered with desquamating epithelium, and do not usually ulcerate except upon the nose. In consequence of the generation and confluence of new nodules, the skin becomes diffusely, and often quite uniformly, thickened. The surface is red, shining, and tense; here and there upon it there are a few spots of a darker red, covered with denser scales of epidermis.

Lupus hypertrophicus also shows a tendency to subside at its point of origin, and to extend into the surrounding skin. In consequence of the retraction of the new connective tissue which forms during cicatrization, the swollen and reddened integument becomes traversed by interlacing, hard, white cords. In the case of lupus hypertrophicus mentioned above, the whole face had become a rigid, callous cicatrix, traversed by varicose vessels, and presented a frightful deformity. The eyelids were everted, the nostrils dilated; while upon the ears the process was only just commencing. It must not be supposed that, in a given case of lupus, one only of these varieties appears, to the exclusion of the others. On the contrary, they usually exist together, one form generally predominating, although traces of the others are nearly always to be found.

Treatment.—In the treatment of lupus our task is a double one. In the first place, we have to remove the neoplasm which is embedded between the fibres of the cutis, or else, by its gradual growth and subsequent retrocession, it will cause great destruction of the skin; and, in the second place, we must endeavor to prevent the formation of

further and similar deposits in the substance of the cutis. With regard to the first point, all surgeons and dermatologists entirely agree that the lupus nodules must be removed or destroyed; but opinions differ greatly as to the surest and most advantageous means and method of effecting this object. It is not for me to go into the merits of purely surgical questions, nor to express a preference for the use of the knife, the actual or the potential cautery, nor to declare in favor of nitrate of silver, or caustic potash, rather than for nitric acid, arsenic, iodide of mercury, or chloride of zinc. In my opinion it matters much less what particular caustic is employed, than that the operator should be well versed in the management of the caustic, and should be familiar with its action. Very obscure and ill-educated surgeons often acquire a reputation from their wonderful success in the treatment of cancer, and of "eating-sores," by means of a secret remedy. It would be wrong to assume that all such reputations are without foundation. Individuals of this class, the so-called "cancer-doctors," by dint of operating daily, and in numerous cases, with one particular kind of caustic, finally become more skilled in its use, and know better how to adapt it to especial instances, than the most distinguished surgeons. Patients, in whom the application of caustic is indicated, may be intrusted to their hands with perfect reliance. But if the state were to buy the secret, science would be but little benefited by the purchase, even though the seller were to act with perfect honesty; for other surgeons operating with the same caustic will not be nearly so successful as the original possessor of the nostrum.

The second indication in the treatment of lupus is far more difficult of fulfilment than the first. Unfortunately, we know of no sure means of preventing the recurrence of lupus nodules in the vicinity of the old site of the disease, nor in the scar of an old lupus, whether healed spontaneously or by artificial means. We are not agreed even as to the necessity of an internal medication for lupus, in addition to the local treatment; or whether we should rely upon external application to produce an alterative effect upon the nutrition of the endangered region. The internal medicines most in vogue are the iodide of potassium, with or without an addition of iodine, and cod-liver oil, which has been given in the enormous dose of twenty-five tablespoonfuls a day. In badly-nourished subjects, and even in those who are not scrofulous, experience warrants the trial of these articles, especially the oil, in very large doses, although not such excessive ones as the above—say six tablespoonfuls a day. In recent cases, and in robust and plethoric subjects, in whom the lupus was making rapid progress, I have seen remarkable benefit obtained from *Zittmann's* decoction. If the disease should come to a stand-still during this treatment, and

for some time after it, and should it break out again several weeks or months after the treatment has been suspended, the same procedure must be repeated.

Among the external applications which have been employed for the purpose of modifying the nutritive state of the skin, and to prevent the recurrence of the eruption, are the ointments of the iodides of mercury, sulphur, and potassium.[2]

VII.—*PARASITES IN THE SKIN.*

The development of microscopic fungi is a constant accompaniment of certain diseases of the skin; in others, again, they are sometimes present, while as a general rule they are absent. *Favus*, *pityriasis versicolor*, and *herpes tondens* belong to the former class. The constant presence of fungi in these diseases, and their contagiousness by transfer of the fungi from one individual to another, is our sole proof that the generation of such fungi is the primary and essential lesion of the diseases. As the fungi are always found, and as transmission of the disease by transfer of the fungi always succeeds, no doubt can be entertained of the parasitic nature of these affections. To our description of favus, herpes tondens, and herpes versicolor, we shall add an account of the itch, as the cause of the cutaneous lesions in this disorder is the presence of a parasitic animal.

The *acarus folliculorum* is also a very common parasite. It exists in most persons, and may easily be obtained by scraping the nose with the back of a knife. As this animal does not give rise to any disease of the skin or its follicles, of which it seems to be a perfectly harmless inhabitant, it possesses no pathological interest, and we may forego further description of it, and omit the account of the various hypotheses which have been advanced as to its importance as a matter of natural history.

CHAPTER XIX.

FAVUS—PORRIGO FAVOSA, LUPINOSA.

Etiology.—*Schönlein* was the first to demonstrate that certain dry, straw-colored crusts, found chiefly upon the hairy scalp, and formerly called *porrigo favosa*, or *tinea lupinosa*, and which were regarded as the product of a dyscratic pustulous inflammation of the skin, really consisted of a mass of fungous spores and germinal filaments (*the oidium, or achorion Schönleinii*). The spores of favus

do not take root very readily; hence the transfer of a mass of such spores from one person to another by no means invariably results in a transplantation of the disease. However, the cases in which a healthy person has been thus infected artificially are numerous enough to place the fact beyond a doubt that the growth of the fungi is no mere accident, but that it is an essential condition of the disorder. Whence the seeds of favus have come, cannot, in many instances, be determined; but I believe that the enormous multitude of sporules of different kinds constantly floating in the atmosphere will always afford opportunity for the implantation of favus, and that such implantation will always take place whenever conditions favorable to the growth of the germ present themselves. Opinions differ as to whether or not the favus-plant is a peculiar species of fungus which only appears upon the scalp and which always develops in the same manner. *Hebra* inclines to the view that the various shapes presented by the skin-diseases due to the presence of vegetable organisms, depend rather upon the stage of development of the plant, upon whether the spores preponderate over the filaments, and upon whether the parasite take root upon the epidermis or upon the hairs, and that it does not depend upon any specific difference of species in the spores themselves. I have this objection to make against this opinion: that, to the best of my belief, complications of these diseases, and transitions of pityriasis versicolor, with favus or herpes tondens, never occur, and that complications of favus and herpes tondens are rare. These facts make it improbable that the differences between these parasitic diseases are mostly due to differences in the situation and stage of development of the same fungus. The most simple explanation of the exceptional cases is, that sometimes the favus and herpes fungi are transplanted simultaneously to the same individual. According to the researches of *Hofmann* of Giessen, who has cultivated the fungus, it is identical with the *mucor racemosus*, and is sometimes accompanied by the *penicillium glaucum*, but only as an accidental occurrence. Dirt seems to be the condition most favorable to the implantation and growth of the germs of favus; at all events, the disease is far more common among the lower classes, whose members often neglect to wash and comb themselves, than among the well to do, among whom the appearance of favus is exceptional.

Symptoms and Course.—The seat of favus is almost exclusively upon the hairy portions of the scalp, and it is rarely found in other situations. At the commencement of the disease we find the affected skin studded with small yellow bodies, scarcely of the size of a pin's head, which lie somewhat embedded in the skin, and each of which is perforated by a hair. These bodies consist of masses of spores, which

nave developed in the funnel-shaped mouth of the hair-follicle, and have elevated the epidermis around the hair. When the masses of favus are numerous, and as they increase in size, they come into contact, coalesce, and finally form a continuous rind of fungi, covering the entire scalp (*favus confertus s. confluens*). If, however, the favus-spots are separate and do not encroach upon their neighbors, a very characteristic formation takes place, consisting of round, concave scabs, like crabs'-eyes, the lower convex surface of which lies embedded in a depression in the atrophied cutis, while their upper surface has elevated edges with a depressed centre (*favus scutiformis*). This thick, dry, sulphur-yellow scab consists of a structureless capsule, which adheres very firmly to the thin layer of epidermis beneath it, and of innumerable filaments and spores. As the hair-follicle and hair itself become overgrown by the fungi, the growth of the hair is impeded; it loses color, and becomes dry, thin, brittle, and falls out. When favus is situated upon a part of the skin destitute of hair, the mass of spores generally becomes loose after a few weeks and falls off, thus putting an end to the disease. Its course is different upon the hairy scalp. Here, too, the duration of the individual masses of favus is limited, but the scabs adhere longer, and the fungi which they contain infect the parts about them. When finally the scab detaches itself, the pressure which it has exerted upon the skin leaves the latter in a state of atrophy which is never repaired. The hair does not usually grow again upon the former site of the disease, and the papillæ which have more especially suffered from the atrophy can only reproduce a thin layer of epidermis; hence, for the rest of the patient's life, there remain upon his head bald smooth depressed spots, whose edges are often surrounded by more recent crusts of favus. The growth of the fungus, the scratching provoked by it, and the lice by which it is often accompanied, frequently cause eczematous and impetiginous inflammation of the scalp. When this complication occurs, besides the favus-crusts, there are other scabs, the desiccated product of the eczematous exudation. These scabs are easily distinguishable from those of favus. Under the latter, the skin, though depressed, is always covered by a thin layer of epidermis; under the former, we always find an excoriation.[3]

Treatment.—Difficult as it is artificially to induce the favus-spores to grow, it is equally difficult to destroy them when once established upon the hairy scalp, and to arrest their further ravages. We have said that the fungi form within the hair-follicles and upon the hairs themselves; hence, not only must all the crusts be removed, but all hairs upon the diseased surface must likewise be extracted. The former object is best attained by saturating the crusts with grease, until

they are completely softened. The head is then to be enveloped in a poultice for some time, after which it is to be carefully washed several times a day, with soap and water applied on a soft brush. For the removal of the diseased hairs, the method formerly in vogue, of covering the head in a so-called "pitch-cap," and, when the hairs had become firmly adherent, of forcibly tearing it off again, is both cruel and uncertain. It is far better to draw them out one by one with a pair of cilia-forceps. After the first few days, this process ceases to seem tedious and troublesome, and, after a few sessions, a quarter of an hour daily devoted to the purpose will be all that is required. Besides the criterion afforded by their dryness, their lack of lustre and faded color, the diseased hairs may be distinguished from the sound ones by the fact that they may be extracted with greater ease and with less pain than the latter. This daily brushing and depilation must be steadily continued for several months, if we expect to cure the favus radically. Tedious and troublesome as this treatment is, yet it is the only one from which we can anticipate any benefit. Other remedies are useless, unless applied in combination with the above procedure; and if so combined, it always is questionable which of the two has really been of service. Even the famous cures of favus, of the brothers *Mahon* in Paris, which consisted in the inunction of a salve of carbonate of soda with lime, and in afterward powdering the head with charcoal, seem to have been effected mainly by careful depilation. The pariciticide remedies most to be depended upon, during and after depilation, are weak solutions of corrosive sublimate (gr. ij–iv to a pint of water or of alcohol), the oil of turpentine, and very dilute creasote.

CHAPTER XX.

HERPES TONDENS.

Etiology.—Not only *herpes tondens*, but most cases of *herpes circinatus*, as well as many of *lichen circumscriptus*, *impetigo figurata*, *pityriasis rubra circumscripta*, and *porrigo asbestina*, depend upon the development of a fungus, the *trychophyton tonsurans*, which breeds between the epidermic cells, converting them into a white dust, and penetrates into the hairs and hair-bulbs, the latter becoming more or less inflamed thereby. In most cases it may be determined positively, by careful search, that this parasitic disease is transmitted by contagion, and that this transmission takes place less often between man and man, than between man and beast, especially the transfer of the parasite from horses and cows, which are quite often afflicted by it, to men who are brought into contact with them.

Symptoms and Course.—All the above-named diseases exhibit great uniformity of appearance; the differences between them are unimportant, and are based solely upon the difference of intensity in the inflammation to which the growth of the fungi in the hair-follicles gives rise. In mild kinds of the disease, papules form, and an exanthema appears, having the character of lichen. In cases of greater intensity, vesicles or pustules arise, accompanied by a herpetic or impetiginous eruption. When there is no real inflammation of the hair-follicles, we have pityriasis rubra, or porrigo asbestina. When the disease is situated upon the scalp, round spots become observable as large as a sixpence and sometimes as large as a dollar, which, unlike alopecia circumscripta, are not quite bald, but are covered with short, broken hairs, and which so much resemble tonsures, that the affection is well entitled to its name, herpes tondens seu tonsurans. Between the short hairs, the scalp is covered with scales. Upon microscopic examination, the roots of the hair are found to be split up like a broom, and between the twigs so formed there is an innumerable quantity of spores and filaments, which spread into the shaft of the hair in wreaths. The same elements are also found in the scales of the epidermis. When the disease is situated upon a portion of skin which is merely covered with a fine down, this is not so apparent, but it is still characteristic enough to place the diagnosis beyond a doubt, without the assistance of the microscope. If a herpes circinatus, a lichen circumscriptus, or an impetigo figurata, be of a well-marked circular form, abruptly distinguishable from the parts about it, when its edge is of a bright red, and studded with nodules, vesicles, and pustules, its centre being paler, smoother, and covered with mealy dust; when each point of eruption is perforated by a hair; and, finally, when it can be ascertained that the patient has been in contact with a man or beast similarly affected, the diagnosis is sufficiently insured. Herpes tondens may recover without medical aid, the growth of the fungi finally exhausting itself, the broken hairs falling out, and the diseased epidermis becoming detached. Upon the hairy regions of the head, however, the disease will exist for a long time before terminating in this way, while upon surfaces void of hair it is usually of but short duration.[4]

Treatment.—Depilation is indicated in the treatment of herpes tonsurans, but, owing to the brittleness of the hairs, the process is by no means easy of accomplishment; moreover, it is often possible to cure the disease with comparative ease without it, by dint of daily and vigorous washing with soft soap, which is always to be followed by an inunction of white-precipitate ointment.

CHAPTER XXI.

PITYRIASIS VERSICOLOR.

Etiology.—*Eichstedt*, of Greifswald, was the first to demonstrate that pityriasis versicolor, which used to be ascribed to a derangement of the hepatic function, is really dependent upon the growth of fungi in the epidermis. In this affection, the filaments are more numerous than the spores. Its very common occurrence among patients with pulmonary disease shows that the skin of such individuals often presents favorable conditions for the implantation and growth of the parasite. Pityriasis versicolor is a very common affection, and often appears upon perfectly healthy persons.

Symptoms and Course.—The most common seat of pityriasis versicolor is upon the neck, back, chest, and arms; but I have never seen it spread from the neck to the face. In recent cases, small, round, yellow spots, which readily peel off, are found upon the affected skin; in cases of longer standing, the spots are larger or have coalesced, forming irregular patches, which often extend over a large portion of the skin. The yellow spots can easily be scratched off by the finger-nail, and by this means, as well as by their ordinary situation, and, still better, by the discovery of the parasite in them, they may be distinguished from chloasma uterinum and from freckles. Pityriasis versicolor often occasions a slight itching. During summer it generally improves somewhat, probably because the perspiration is freer; but it scarcely ever disappears unless judiciously treated.

Treatment.—I used formerly to treat pityriasis versicolor with a mixture of carbonate of potash ʒ vj, liquor hydrarg. nitric. oxydul. ʒ iij, and aquæ rosarum ℥ vj, which must be well shaken, that the sub-oxide of mercury may not remain at the bottom of the bottle. Under this application, made morning and evening, pityriasis versicolor has nearly always disappeared in a week or ten days. Recently I have found that the same object may be attained with equal facility, by inunction of green soap twice a day.

CHAPTER XXII.

SCABIES—ITCH.

Etiology.—The itch is an inflammation of the skin characterized by the formation of nodules, vesicles, and pustules, and caused by the presence of an animal parasite, the itch-mite, *acarus scabiei*, or *sarcoptes hominis*. The parasite is about $\frac{1}{6}'''$ in length, and has a breadth

of about $\frac{1}{7}'''$, and is recognizable as a round white body to the naked eye. Under a sufficient magnifying power, it is seen somewhat to resemble a tortoise in shape; its convex back is marked with curved parallel transverse stripes, and is armed with spines of varying length. The young insect has six, the adult eight, articulated feet, the anterior pair of which are provided with suckers. In the female, two pair of the hindmost of the feet terminate in long bristles; in the male, only the first of them has bristles, while the posterior pair are also provided with suckers. The head of the mite, which is armed with two horny jaws, separated by a cleft, projects from between the anterior pair of legs. The male insect is smaller than the female, and much less numerous; it is provided with a penis shaped like a horse-shoe; the female, when pregnant, embeds itself between the lamellæ of the epidermis, and forms a burrow which is often several lines in length, and sometimes an inch or more. These passages contain empty shells of the mites, together with their blackish fæces, and their eggs in every stage of development. The burrows dug by the males are shorter, and therefore more difficult to find. The eggs seem to mature in about eight days. The young mites, after escaping from their shells, abandon the gallery of their mother, and burrow for themselves in the vicinity. After casting their skin repeatedly (twice at least), and acquiring an additional pair of legs with the first moulting, they come out of their holes and pair—generally during the night, when the skin is warm, in the bed. Sometimes the male insect visits the female in her burrow. If a fecundated female be transferred from the skin of one individual to another, the latter also becomes infected by itch. As there is no opening in the galleries, and as those in which the pregnant females lodge are stopped up by the eggs and fecal masses, it may be assumed that the female only comes to the surface when her burrow is scratched open. Sleeping with a person afflicted with itch is, therefore, an especially dangerous procedure, although a very brief pressure of the hand will suffice to transfer a mite scratched out of her abode. It is not known how long the insect is capable of living after it has been removed from its natural habitation and feeding-ground; but it seems that those which get into the clothing and linen soon die. One of *Hebra's* discoveries proves the truth of this most strikingly, and indeed better than the most minute observation of a few captured mites. In Vienna, where fifteen hundred itch-patients are treated annually, the number of relapses does not amount to one per cent., although their linen, and other clothing, is not steamed nor baked in the louse-oven, nor subjected to any other particular process, in order to kill the itch-mite and its eggs. We are indebted to *Eichstedt*, *Fürstenberg*, *Gudden*, and others, for our more intimate acquaint-

ance with the habits of the acarus scabiei, a knowledge which has completely overthrown the ancient errors regarding the origin of the itch.

Symptoms and Course.—Generally speaking, a patient is first made aware of his having scabies by an intense itching, which is worse when he is warm in bed, and which is by no means limited to the regions most frequented by the acarus, namely, between the fingers, in the bends of the joints, upon the genitals, and between the nates; for it is also very severe upon the legs, abdomen, and chest. The most conspicuous objective symptoms are nodules, pustules, and vesicles in variable number, occupying the regions mentioned. In very little children, whose head is often kept buried in bedclothes, they are sometimes, although rarely, found upon the face. These nodules, vesicles, and pustules do not denote different kinds of itch, but rather are indicative of the stage of dermatitis excited in the skin by the presence of the parasite, and still more by the scratching. In children, and in other persons with tender skin, the inflammation is most apt to be so severe as to form pustules, and even to suppurate. These nodules, vesicles, and pustules are often excoriated by the nails, and converted into bloody scabs. A much more characteristic sign of the disease is afforded by the burrows of the itch-mite, which, with a little care and practice, are not hard to find. They consist of dotted, sinuous, or zigzag seams, which look like the scars of needle-scratches. At their beginning—that is, at the place at which the insect embedded itself—there is often a vesicle, more rarely a papule or pustule; at its termination there is a somewhat larger blackish or whitish point, corresponding to the actual position of the insect. The burrows are most numerous between the fingers, and upon the inner surfaces of the wrist and elbow-joints, and upon the penis. In order to catch the acarus, a needle must be thrust into the beginning of the burrow, and carefully pushed toward its end, at the same time ripping open the covering. A minute white point, which, upon attentive examination, will be seen to change its position slowly, and which, after this procedure, is generally found on the point of the needle, is the acarus. Not unfrequently an egg may be seen within her, which may be squeezed out of her body by gently pressing upon the fine glass cover. The itch-tracks found upon the penis are always accompanied by infiltration of the skin beneath them. When the insects are excessively numerous, instead of the discrete efflorescences, there is a diffuse eczematous dermatitis, whose scabs are inhabited by legions of mites. This form, which is rare with us, but which used to be of frequent occurrence in Norway, is "Norwegian itch," or Borkenkratze.

Scabies never gets well spontaneously, but is very amenable to treatment; hence it is an innocent disease. No educated physician now believes in "metastasis of the itch" as a result of "incautious suppression" of the malady. Even the homœopaths—in spite of the anathemas hurled by *Hahnemann* against suppression of the "psora" by local applications—now prescribe green soap and sulphur, regardless of consequences. However, notwithstanding the ease with which it is cured, the itch is an affection greatly feared by the so-called educated classes, by whom it is regarded as a plebeian and disgraceful disease. This prejudice is so great, that their horror over the discovery that they have the itch is by no means diminished by the information that it is more harmless and easier to cure than almost any other cutaneous disorder.

Treatment.—To cure the itch, it is only necessary to kill the insects and their eggs, or to remove them from the skin. This done, the eruption induced by the parasites, and by the scratching which their presence provoked, soon subsides. There are many methods of curing the itch with certainty; of one, however, it is claimed, that it affords the most rapid possible relief; that it is less irksome to the patient, and that it costs less, at all events in hospitals. The remedies most commonly employed are green soap and sulphur. When green soap alone is used, every suspected part of the skin must be rubbed with it twice daily, until a decided eczematous dermatitis sets in. With the exfoliation of the epidermis consequent upon this eczema, the itch-mites and their eggs are also got rid of, as the burrows never extend down to the cutis. This cure is a certain one, but it requires from six to eight days for its accomplishment. Meantime, the patient must not wash himself nor change his clothing, and must remain in a room heated to a temperature of 75° to 80° Fahr. He must not bathe until the sixth or eighth day, when the cure is complete. This treatment is not adapted for private practice, being too irksome and unpleasant. The object may be effected somewhat more quickly by using a mixture of two parts of green soap to one of powdered sulphur, which is to be rubbed in as directed above. The English method also consists in an application of green soap and sulphur, with certain additions. The treatment is commenced by a warm bath, after which, for two or three periods of twenty-four hours, the patient is to remain packed in woollen blankets at a temperature of 90° or 96° Fahr. After the first twelve hours, one-third of the ointment (consisting of sulphur ℥ j, white hellebore ʒ ij, nitre gr. x, soft-soap ℥ j, lard ℥ iij) is to be rubbed into all suspected regions; after the second twelve, a second portion is to be applied; and in twelve hours more the remaining third. Twelve hours after the last inunction, the treatment terminates

by a bath. This method, also, though sure and rapid, is unfit for private practice, being inconvenient and disagreeable, and because patients do not like to sacrifice a pair of woollen blankets to the itch. *Wilkinson's* ointment, which contains tar and chalk in addition to the soft-soap and sulphur, is still less adapted for treatment of private patients. For this purpose the simple ointments of lard (℥ j), and sulphur (℥ ss), are more suitable, as is also *Helmerich's* ointment, consisting of carbonate of potash ʒ ij, sulphur ℥ ss, and lard ℥ ij. After one or two initiatory warm baths, the ointment is to be rubbed twice or thrice daily over the entire body, excepting the face, or, at all events, over every spot where any eruption or traces of itch can be found. By this means the disease can be cured with certainty in from eight to fourteen days. When the patient cannot conveniently absent himself from his business, let him take a warm soap-bath at night, then rub the suspected spots, or, better, the entire body, excepting the face, with *Helmerich's* ointment, and in the morning let him take another bath. This procedure also cures the itch in a few days. *Helmerich's* ointment is likewise employed in *Hardy's* "speedy cure." This consists in half an hour's rubbing with green soap, an hour's bathing, during which the rubbing is to be continued, followed by half an hour's inunction with the ointment. The cure is perfected in a few hours, but, though often successful, is not quite sure. The *Vleminckx's* solution, already described, would be a remedy preferable to any of the above-mentioned, being both simpler, more cleanly, and cheaper, were it not that it often produces severe and obstinate eczema in persons with delicate skins. The directions for the use of *Vleminckx's* solution, as practised in the Belgian army, are as follows: Half an hour's bathing, half an hour's rubbing with green soap; then, half an hour's rubbing with the solution of lime and sulphur; then, another bath for half an hour, when the treatment is ended.

Under the impression that it is sufficient to kill the mites and their eggs, and, that if this be done, it is unnecessary to open the burrows, and to effect the removal of both insects and eggs, by the production of an artificial eczema, it has been proposed to rub the skin with parasiticide substances, capable of easily penetrating the epidermis, and especially to use petroleum and balsam of Peru. Nothing can be said against the correctness of the theory; and a series of experiments, by rubbing in these articles, has succeeded. Nevertheless, according to experimenters who have tested the treatment fairly, its effects are uncertain; and it has not superseded the old treatment in many of the hospitals where it has been tried. It would only be advisable to have recourse to Peruvian balsam, or (when that is beyond the patient's means) to try the efficacy of petroleum, when the use of sulphur and

soft soap is contraindicated. It used formerly to be thought necessary to expose the clothing to a temperature at which albumen will coagulate, in order to kill the insects and eggs lodged in them. The linen was boiled; and the cloth garments were baked in the so-called "itch-oven," or sent to the "kettles" at the establishments for cleaning feather beds, which exist in all large cities. We have already mentioned that *Hebra* declares this to be superfluous. According to the undisputed testimony of this authority, the treatment of the itch in private houses is much less difficult, and promises far more success than was formerly supposed; and the danger of catching the disease anew from clothing, bedding, furniture, or other utensils that have not been disinfected, is purely imaginary.[5]

VIII.—*DERANGEMENTS OF SECRETION IN THE SKIN.*

We are aware that gaseous, watery, and oleaginous secretions are given off from the surface of the skin. There is no doubt that, under certain conditions, the quantity and quality of these secretions vary from the normal; but we are imperfectly acquainted with the nature of these variations, and with the sources whence they arise.

The secretion of gases, especially of hydrogen gas, is increased in febrile disease—apparently from causes purely physical—whenever there is much elevation of the temperature of the skin. If we weigh a patient who has had a fever, we shall find him much lighter than he was prior to his illness. If we take account of every thing eaten and drunk by him, and if we deduct from this the loss of weight, and the weight of the fæces, sputa, urine, etc., which have been evacuated during the sickness, it will be found that the excretion through the skin (and lungs) greatly exceeds the normal amount. It is well known that an increase of the insensible perspiration is not always accompanied by an augmentation of the secretion of sweat; and indeed, that the skin usually is dry while the former is going on most rapidly. A copious supply of watery liquid greatly increases the amount of gaseous perspiration. An athletic medical student, for the sake of experiment, dieted himself for a long period of time upon exactly the same quantities of food and drink as were given to two patients with diabetes mellitus. The quantity of urine which he secreted was much increased, but upon calculating the daily amount supplied to the system, and upon deducting the weight of the daily excretions of urine and fæces, as well as the gain in weight of the body, from the weight of the food and drink which he consumed, it was found that the insensible evaporation from the skin and lungs had been very considerably

augmented. There had been no profuse sweating; and it is very probable that, besides the conditions mentioned above, there are others which likewise serve to increase the imperceptible perspiration.

A diminution of the insensible perspiration is probably of common occurrence, for instance, in a very cold or very moist atmosphere; but hitherto, this fact has only been demonstrated directly by weighing, in cases of diabetes mellitus.

We know nothing positive about the qualitative changes in the cutaneous evaporation, and can merely suppose that in febrile disorders this excretion not only increases in quantity, but is altered in its composition. That such a change does take place seems to be indicated by the peculiar odor emitted by the gaseous perspiration of patients suffering from the acute exanthemata.

The secretion of sweat is much increased in most people when they are exposed to certain influences. Both healthy persons, and persons suffering from chronic diseases free from fever, if weighed before and after a course of profuse artificially-induced sweating, are found sometimes to have lost four or five pounds' weight in a few hours. We have no precise knowledge of the conditions which regulate the production of sweat. Healthy persons sweat profusely when covered by blankets and made to drink copiously; but in most febrile diseases they do not perspire at all, though treated in the same manner; while there are other disorders attended by fever (not merely the inexplicable sudor angelicus of the middle ages, but many cases of pneumonia and rheumatism), in which the sweating is profuse throughout the whole attack. That the secretion of sweat, like that of the saliva, is dependent upon nervous influence, is evident from the effect which the mental emotions exert upon its production. Some persons perspire more readily and profusely than others. An excessive tendency to perspiration is called *hyperidrosis*. It generally depends upon unknown causes; but in some instances it unmistakably is a consequence of repeated artificially-induced diaphoresis. *Bärensprung* mentions many examples of hyperidrosis proceeding from the above cause. I myself have never noticed any great increase in the secretions of the skin at the commencement of a course of diaphoretics, and have only observed it in persons who had been under treatment for some time, and hence can confirm the assertion that profuse sweating augments the tendency to diaphoresis. When the secretion of the sudoriferous glands cannot escape, owing to obstruction of the gland-ducts, or because the latter are incapable of transmitting all the secretion formed, it emerges around the sweat-duct under the epidermis, and, lifting the latter, forms small, clear vesicles containing an acid liquid, which are called *sudamina*. Obstruction of the efferent ducts of the sudoriferous

glands occurs most frequently when the secretion of sweat has been arrested for some time; for instance, in the first weeks of typhus. Sudamina, also called *miliaria alba*, have nothing in common with the *miliaria rubra*, excepting that the source of both affections is in the sudoriferous glands. Miliaria rubra has nothing to do with retention of perspiration, but consists in an inflammation and exudation, induced by excessive diaphoresis; and for this reason we have classed miliaria rubra under the eczematous affections. An eruption of sudamina has no influence upon the course of the disease which it accompanies, and is seen as often in critical sweats as in the profuse perspirations which break out in unfavorable phases of a disease, and even in that stage which immediately precedes dissolution. The profuse sweats of the miliary fevers of the middle ages could hardly have been of a critical character, as they appeared when the disease assumed a malignant form; hence the miliary eruption was considered an unfavorable sign.

A partial hyperidrosis, confined to the palms of the hands, the soles of the feet, to the armpits, and to the region of the genitals, is of far more common occurrence than general hyperidrosis. The "sweaty hands and feet," by which many people are troubled, are unnaturally cool. The disgusting odor emitted by the perspiration of the feet and armpits is not dependent upon the presence of ill-smelling ingredients in the secretion, but upon putrefactive decomposition of the perspiration, sebaceous matter and macerated epidermis of the skin. *Hebra*, undoubtedly, goes too far in asserting that this offensive decomposition only takes place in old shoes saturated with sweat, and that people usually accused of having stinking feet really have stinking boots. This offensive perspiration was formerly regarded as a wholesome excretion, to be encouraged and cherished; and many complaints were ascribed to its repression. Such views nowadays have been declared altogether unfounded, upon the high authority of *Hebra* and others; and indeed, to say nothing of the fact that it is by no means easy to check sweating of the feet, its arrest immediately before the outbreak of a fit of illness is, in most cases, the result of the illness and not its cause. But I think that it is saying too much to deny absolutely that such suppression of perspiration may possibly now and then be the cause of disease. There is no doubt that people are often made ill by exposure to a degree of cold while perspiring, which would have done them no harm had the skin not been moist with perspiration; and it is equally certain that diaphoresis artificially induced, often is of the utmost benefit in diseases caused by exposure to cold. Admitting the correctness of these facts, it is not absurd to suppose that sometimes the chilling of perspiring feet may induce disease, nor that the reëstab-

lishment of the perspiration may act beneficially upon disease so induced. The utmost objection that can be made against the statement, that "suppression of the perspiration has caused illness," is, that it is too indefinite; since we are quite ignorant as to why it is that the chilling of a perspiring body so often gives rise to disease. Hence, I consider it to be entirely warrantable, when it has been proved, or even when it is very probable, that the cessation of an habitual hyperidrosis of the feet has been followed by sickness, that we should attempt, by means of hot stimulating foot-baths and by warmly clothing the feet, and by sprinkling the stockings with flour of mustard, to reëstablish copious diaphoresis of the extremities. I do not mean by this, that persons suffering from offensively-perspiring feet and armpits are to be forbidden to wash and bathe, or to use cold water for the purpose. On the contrary, the judicious use of cold baths and of cold washing, so far from being hurtful, is of the utmost benefit to persons who perspire too freely, and who are liable to take cold. In fact, I think it probable and by no means inconsistent with the opinions expressed above, that the cautious checking both of local and general hyperidrosis is the best means of obviating the danger of catching cold by which it is accompanied. At all events, people with sweating feet should frequently change their shoes and stockings. When the epidermis becomes soft and pasty from maceration, and when it inclines to peel off, exposing the rete Malpighii, making it painful and difficult for the patient to walk, *Hebra* recommends, as an excellent remedy, that the soles and toes should be smeared for some days with the unguentum diachyli, and then be wrapped in linen rags. He claims that, by this treatment, not only does a new and firmer layer of epidermis take the place of the old and softened one, but it causes a diminution of the hyperidrosis for some time afterward. I have no experience of my own testifying to the efficacy and harmlessness of this procedure.

We have already mentioned that a diminution of the cutaneous secretions may form one of the symptoms of senile marasmus. In other instances, as has already been mentioned, it depends upon derangement of innervation; in others, again, it is due to disease of the skin, to psoriasis or to ichthyosis, while, finally, cases exist of *anidrosis* and now and then even anidrosis of half the body, the causes of which are quite unknown.

With regard to the qualitative alterations of the perspiration, the presence of biliary pigment in the sweat of persons with icterus, upon which the yellow stain of their clothing depends, is perhaps the best known. *Bärensprung*, however, believes that the vehicle of the pigment is not the liquid of the perspiration, but the epithelial cells which

it contains. The composition and source of other substances, which sometimes appear in the sweat and which likewise stain the linen, are unknown. In a few instances of suppression of urine, trustworthy observers have discovered crystals of urea upon the skin.

In some persons, the sebaceous secretion of the skin is so much increased that the hair and cutaneous surface, especially that of their face, always shines as if it had been freshly anointed with pomade or grease. In others it is so deficient that, unless replaced by an unguent, the skin and hair are always dry, dingy, and disposed to crack. The former condition is most likely to exist in persons with an exuberance of fat throughout the entire body; the latter in individuals with little fat, or in those who are suffering from wasting disease.

Qualitative changes in the sebaceous secretion are often observed, in which the unctuous matter of the skin does not remain liquid, but assumes a more solid consistence. *Bärensprung* believes that the increased solidity of the cutaneous secretion is due in part to a preponderance of solid fat, and in part to an admixture of a larger amount of cast-off glandular epithelium. The increased solidity of the sebaceous matter of the skin is usually accompanied by exuberance in its quantity, thus in some measure justifying the name *seborrhœa*, applied to a condition of the skin in which it is covered with crusts of dry sebaceous matter. Seborrhœa is most frequently observed upon the scalps of infants, where the inspissated secretion, rendered brown by admixture of dust, often forms scaly crusts of nearly a line in thickness, of which superstition forbids the removal; and it is not until the second year, when the scabs are lifted from the scalp by a freer growth of the hair, that they break up into separate scales, and are removed as such with a comb. Many a case of the so-called pityriasis capitis, in which there are many white scales, some of which adhere to the skin, some sticking to the hairs, while still others are sprinkled upon the clothing, are the result of seborrhœa. Microscopic examination, by which it is found that the scales contain numerous oil-globules, affords the best means of distinguishing this form of seborrhœa from a dry eczema capillitii. Finally, thick crusts of sebaceous matter, browned by admixture with dust, and whose surface is generally broken up into numerous blocks, appears upon the cheeks, nose, eyelids, ears, and nipples, as well as upon other parts of the skin where a copious lanugo prevents the fall of the crusts. Abnormal secretion of sebaceous matter may go on so rapidly, that the cast-off scabs are speedily replaced by new ones, which greatly disfigure the patient, who is usually a female suffering from menstrual derangement (*ichthyosis sebacea*, *Rayer*). Thick, hard crusts of sebaceous matter are to be softened with liquid fat, and then removed with caution, since the epithelial coat beneath them is ex-

tremely tender. Systematic washing with soap and water, or alcohol, is the best preventive of the formation of new crusts, although it does not alter the abnormal character of the cutaneous secretion. For seborrhœa capillitii the hair-dressers use an evening lotion of "honey water," and a morning inunction of the head with macassar-oil. This is a most rational procedure; for the honey-water, which contains alcohol, loosens the scales, and the admixture of liquid of Macassar-oil to the solidifying sebaceous matter prevents it from drying up into new scales. Instead of the honey-water, however, any other liquid containing alcohol, such as French brandy, or eau de cologne, may be employed, and any other oil will do instead of Macassar-oil. In treating *Rayer's* ichthyosis sebacea, especial attention must be paid to the state of the sexual organs, since experience teaches that, upon relief of any existing derangement of the latter, the tendency to excessive production of sebaceous matter also subsides. Local applications alone merely prevent accumulation of the secretion, but not its superabundant generation. When, instead of flowing from the follicle, the secretion solidifies within it, creating hard masses like the shell which forms on the scalp in seborrhœa (as above described), we have the *comedo*, the *milium*, or *atheroma*. In a *comedo*, the outlet of the follicle is obstructed by a plug of black, hardened sebaceous matter, mingled with dust. Upon compressing the follicle, its contents are squeezed out of the contracted orifice, and, like any other pasty substance, forced through a sieve, it assumes a fusiform shape. A *milium* means a follicle distended by hardened secretion to the size of a millet-seed, and covered by epidermis, so that there is no visible black speck in its middle. *Atheromata* are sebaceous glands distended by their inspissated contents to the size of a hazel-nut, walnut, or pigeon's-egg. It is easy to understand that an excessively distended sebaceous gland blends with the hair-follicles, into whose outlet it opens. In treatment of the comedo we can merely remove the accumulated matter, it being out of our power to prevent its production. For persons who are vain enough to submit to a somewhat unpleasant process, in order to rid themselves from time to time of their comedones, I employ the following prescription (originally proposed by *Richter*), and have always obtained very good results from it, if not very speedy ones: A mixture is to be made of rye-meal, honey, and yeast; after waiting until it ferments, the fermenting paste is to be applied at bed-time upon the part of the face upon which the comedones are situated. Next morning, those comedones which evidently are somewhat loosened and elevated—but only those—are to be pressed out, not between the nails, or under a watch-key, but under a gentle pressure of the fingers. This process must be kept up for several weeks. We

have already mentioned the beneficial effect of the preparations of sulphur, when treating of acne. For the treatment of milium, *Hebra* recommends removal of the epidermic covering by bathing it with a moderately strong solution of caustic potash (one drachm to a pint of water), or by the application of soft soap upon a flannel rag. Treatment of atheroma consists in division and extirpation of the degenerated follicle.

[APPENDIX TO DISEASES OF THE SKIN.

SCLERODERMA.

Scleroderma occurs at any age, but especially in middle-aged women from unknown causes, and is due to a gradual increase and atrophy of the cutaneous connective tissue especially of the face, arms, etc., which becomes hardened, pale, red, or pigmented, and adherent to the parts beneath, so that the lineaments of the face may be stiffened, the lips and nostrils shortened, the fingers stiff, etc. The disease goes on for years, and when arrived at the stage of atrophy is incurable ; but earlier it may subside. This favorable result does not appear due to rubbing, bathing, mercurial salve, or the internal use of iodide of potash ; but tonic treatment and good nourishment may do good. The nature of this disease is not fully understood ; while some attribute the changes in the skin to stasis of lymph, either from thickening or from disease of the lymphatics (and regard it as related to elephantiasis), other observers, who have seen the joints and some bones implicated, refer the disease to trophic neurosis originating in the ganglionary system. A different form of the disease is sclerema neonatorum, or cellular-tissue hardening, which attacks feeble, premature children under fourteen days old. There is decided disturbance of circulation in the outer portions of the body and great depression of the temperature ; the skin begins to harden on the legs, and this hardening extends to the trunk, arms, and even face. The portions of skin affected are sometimes œdematous, and subsequently hard as boards, cold and cyanotic ; the stiffness greatly hinders all motion, the face is wrinkled, sucking and drinking are impaired, the bodily temperature falls markedly, and death usually occurs within a week. On autopsy, besides œdema of the skin and perhaps of the deeper parts, the fatty tissues are found unusually firm ; some regard this as due to the sinking of the bodily

temperature. But generally there are remains of other morbid affections, which have existed with or before the skin trouble, and must have impaired the circulation, as pulmonary atelectasis, pneumonia, heart-disease, intestinal catarrh, and ulcers.

Treatment is to be directed against these causal diseases, to securing good nourishment by a wet nurse, *Nestle's* food, nutrient enemata, wine, ether, musk, etc., and improving the circulation by wrapping the child in flannel, hot baths, etc.

LEPROSY—ELEPHANTIASIS GRÆCORUM.

Leprosy, which was formerly so dreaded, and which is still endemic in some countries, and was so even in Europe previous to the fifteenth century, still occurs sporadically. Although our knowledge of this disease has of late been greatly enriched, its origin is still uncertain; but it has been ascertained that a minority of cases were hereditary, and that the disease is not contagious. Although the symptoms of leprosy are not always the same, still the cases show that they are of the same general character. Among the forms that have been described are *lepra maculosa*, *l. tuberculosa*, and *l. anæsthetica*. The disease rarely begins before the seventh to the tenth year; it is more common in youth and middle age. The local symptoms are usually preceded for a long time by general symptoms, such as heaviness, chills, loss of appetite, vague pains, evening fever, etc. In the maculous variety, spots come on the skin; these may disappear on pressure, and they may be dark or white; the disease may run its course with these appearances, or combine with the other two forms. In the tuberculous form, sensitive nodules as large as a nut form on the maculæ or on healthy skin. These nodules may be scattered or congregated; they come especially about the face and limbs. After it has lasted a long time, the mucous membrane of the mouth, larynx, nose, and eye may be affected. Leprous nodules may last for years and then pass away, leaving a dark pigmentation or spot of white atrophy in the skin; more rarely they soften and form abscesses, or ulcerate, even going deep enough to expose the bones and open the joints, especially of the hand and foot.

Although cutaneous anæsthesia accompanies spotted and nodular leprosy, in the form called anæsthetic loss of sensation exists in the skin without spots or nodules. Later the anæsthetic spots become flabby, yellow, and atrophied; the muscles of the face and hands participate in the atrophy; the skin breaks through, leaving a torpid, lifeless opening, which slowly enlarges, and finally exposes or even detaches phalanges or portions of limbs. The anæsthesia of

the skin and the trophical changes have long directed attention to the nervous system, and large nerve-trunks have been found nodulated. But in the central nervous system also changes have been found, and perhaps more perfect methods of examination will there find the causal changes. In one case *Langhans* found great softening of the gray commissures and posterior horns, showing the spinal cord to be the seat of disease in that case at least. The neurotic nature of leprosy is rendered more probable by the frequent coincidence of pemphigus (which is regarded as a neurosis), and by its tendency to occur symmetrically on the two sides.

The prognosis is bad. Though perhaps not for years, it usually causes death, preceded by loss of the limbs, face, etc. Sometimes the course is milder, and after many years ends in recovery.

Treatment is of little avail. None of the vaunted specifics have held their own. So far as at present known, the best treatment is change of residence, good hygienic surroundings, cleanliness, nourishment, fresh air, iron and cod-liver oil, and attention to special symptoms. At a leper asylum in British Guiana gurjun oil is said to have proved beneficial, as has also chaulmoogra oil, in doses of five to twenty drops three times daily, while it was applied externally combined with psoralea corylifolia.]

ADDITIONS TO THE REVISED EDITION OF 1880.

DISEASES OF THE SKIN.

1.—P. 455.

Besides ordinary erythema nodosum, which occurs in children and youth otherwise healthy, *Uffelmann* describes a modified form with certain constitutional peculiarities, where the patients have an hereditary tendency to tuberculosis, feeble health, and delicate frames.

Bohn regards erythema nodosum (as well as peliosis rheumatica) as an inflammatory infarction from embolism of the cutaneous arteries, while other observers make a decided distinction between cutaneous embolism as seen in endocarditis and exudative erythema proper.

Lewin defines exudative erythema as a vasomotor neurosis, and calls attention to its symmetrical occurrence on the two sides of the body. It is not unfrequently so modified as to resemble other diseases. Pustules forming on the erythematous spots may induce the

appearance of variola ; or it may be accompanied or followed by articular inflammations, giving a resemblance to articular rheumatism ; it is even possible that some of those cases of valvular disease of the heart of obscure origin may have been related to a precedent erythema exsudativum. Finally, *Lewin* believes that in women the erythema may be due to inflammation of the urethra ; that is, it is the manifestation of an irritation starting from the urethral mucous membrane and reflected to the vasomotor nerves of the skin, as gonorrhœal rheumatism is in males.

Another form has been observed as an epidemic in Paris, the Crimea, Mexico (acrodynia), as well as that called pellagra in Italy and the South of France.

2.—P. 505.

The use of even nitrate of silver requires some practice. Each lupus nodule should be bored into with a sharpened point, and loosened up from the sound skin ; which is not difficult, as the former is readily broken up, while the latter is resistant. The pain induced ceases after a few hours. The operation may be repeated once or twice a week ; it would be too painful to destroy all the nodules at once, where the nodules are numerous and disseminated. *Hebra* and *Kaposi* recommend a sort of weak *Cosme's* paste (arsenic. alb. gr. x, cinnabar. ʒ ss, ung. aquæ ros. ʒ ss), which is spread on muslin and applied for three days, being renewed every day. The second day there is mild, the third day tolerably severe pain, and œdematous swelling of the face and eyelids ; but the lupus nodules have become dark-brown sloughs, which are detached by suppuration after a few days, leaving cavities which soon granulate. If the nodules have already ulcerated, it may suffice to apply the plaster for one or two days. Arsenical poisoning rarely if ever occurs from the above treatment. Another plan is to scarify the nodules at intervals of some weeks. *Volkmann* recommends scraping out the nodules with a sharp scoop, and if necessary subsequently scarifying or cauterizing the raw surface ; this operation is painful, and requires anæsthesia ; the same is true of treatment by the galvanocautery.

But destructive treatment is not always required. Where instead of nodules there is merely infiltration of the skin, resolution may take place without ulceration ; so we may try to aid resolution by irritating the part with concentrated solution of nitrate of silver or tincture of iodine with intervals of rest. Mercurial ointment may be applied daily.

3.—P. 507.

The microscope shows that the favus bodies consist of an epidermis coating, containing fungi and fine granular cement. From the surface of the capsule numerous fungous filaments project into the favus body. At first these filaments are simple, but deeper they become branched and finally form chains. The same fungous elements are found in the roots of the hairs and in the hairs themselves.

4.—P. 509.

We should not confound herpes tondens capillitii with the disease known as alopecia areata. In the latter, which occurs both in children and adults, the hair falls from a circumscribed spot in the midst of a luxuriant growth, leaving circular bald spots, which may unite with other similar spots. But alopecia areata is not due to fungi ; it is a trophic nervous affection. The bald spot is not red or covered with scales, and after some months the hair grows again without treatment.

Herpes tondens might be mistaken for roseola syphilitica or psoriasis ; but in the syphilitic affection there is no scaly deposit ; in psoriasis, on the contrary, the red spots are covered with thick regular scales.

5.—P. 515.

A treatment for scabies, recently again resorted to and highly spoken of, is with balsam of Peru, which when brought in direct contact is said to kill the insect in 20 to 40 minutes. The patient is to have a warm bath, then to rub the whole body except the head, but more especially the parts most affected, viz., the hands, feet, large joints, penis, scrotum, breasts, and nates. The treatment requires about one ounce of balsam, which may be used in four to six applications, made within two days and without changing the underclothes ; then wash the body well. This treatment has the advantage of not inflaming the skin or being so disagreeable as most others. A similar but somewhat less expensive treatment is by using petroleum 30 parts, olive oil 8 parts, in the same manner.

DISEASES OF THE ORGANS OF LOCOMOTION.

In the present section we would briefly call attention to all the diseases of the joints, bones, or muscles coming within the reach of internal remedies, whether they be idiopathic affections of these organs, or the prominent symptoms of constitutional disease, of a cachexia or dyscrasia.

RHEUMATISM.

The series of morbid processes which were formerly grouped together as rheumatism has grown smaller, it is true; all those diseases of the mucous membrane, and in the parenchymatous organs, which were formerly called rheumatism, have been excluded; and we have also ceased to designate as rheumatic those cases of inflammation of the pleura, pericardium, and peritonæum, referrible to cold, or for which we can find no cause; but still the number and variety of morbid processes classed under the head of rheumatism are sufficient to render a precise definition of the term impossible.

We may mention the following points as characteristic of the forms of disease at present considered as rheumatic: 1. The seat of the disease in the fibrous tissues, the joints, aponeuroses, sheaths of the tendons, neurilemma, periosteum, or in the muscles and tendons; 2. The painfulness of the affection, which is due in many, probably in all cases, to stretching of and pressure on the elements of the tissue by the dilated capillaries and an inflammatory œdema; 3. The absence of traumatic causes and a kind of independent occurrence of the disease, that is, its independence of other acute and chronic diseases. I think that the negative description given in 3 is more distinctive than a positive one would be in its place, which would perhaps refer the origin of the affection to catching cold and to atmospheric influences. Frequently as catching cold and atmospheric influence lead to rheumatism, it is not at all proved that they are its sole cause. In practice, all painful inflammatory affections of the above-named tissues that oc-

cur primarily and idiopathically, and are not of traumatic origin, are termed rheumatic, no matter whether the disease can be referred to catching cold or not. Another characteristic of rheumatic affections is the frequent disproportion between the severe subjective symptoms and the insignificant anatomical changes, as well as the tendency of the disease to pass from the part first affected to others of analogous structure and function. But the proportionately severe pain in rheumatism is less due to the variety than to the location of the disease; at least traumatic, gouty, and other inflammations of moderate degree, are very painful, when they attack fibrous tissues. Nor is the fugacity of rheumatic affections, their tendency to shift position, entirely characteristic, for no one would object to terming rheumatic an idiopathic, painful affection of a joint, which did not show this fugacity, but remained limited to the same joint for years.

CHAPTER I.

ACUTE ARTICULAR RHEUMATISM—RHEUMATISMUS ARTICULORUM ACUTA—RHEUMARTHRITIS ACUTA—FLYING GOUT.[1]

In acute articular rheumatism the synovial capsule of a greater or less number of joints is the seat of an inflammatory disturbance of nutrition. The simple name of polyarthritis acuta would be enough for this disease, if there were not a secondary polyarthritis acuta occurring in the course of infectious diseases from which acute articular rheumatism is to be distinguished as polyarthritis acuta rheumatica seu idiopathica. In most cases of acute articular rheumatism, the inflammation of the synovial capsule does not attain a very high grade. The exudation into the joint is usually neither copious nor fibrinous, nor does it contain many pus-cells. The swelling visible about the affected part depends mostly on inflammatory œdema of the connective tissue around the joint. But this slight degree of inflammation and the quality of the exudation are by no means constant peculiarities of the disease; on the contrary, there are some cases where the inflammation has attained a far higher grade, and where a fibrinous or purulent exudation is deposited.

The predisposition to acute articular rheumatism is very unequal in different persons, without our knowing the reasons for this dissimilarity. Some persons seem to inherit a predisposition to the disease. Those who have had one or more attacks are very liable to it. The disease is rare in early childhood and in old age; it occurs most frequently between the ages of fifteen and forty years. Men are attacked as often as women, or perhaps more frequently. The predisposition appears to

be somewhat stronger in robust, full-blooded persons than in those who are weak and anæmic.

Among the *inducing* causes of acute articular rheumatism the more important are temporary exposure to cold, either by suddenly wetting the heated body, or by being subjected to a draught, and continued residence in damp dwellings and working in damp places. The working-classes, who are chiefly exposed to these bad influences, are consequently more subject to the disease than the well-to-do classes. In most cases the exciting cause cannot be recognized. The disease occurs all over the world, but is more frequent in the temperate zones than in the polar regions or the tropics. At some times, particularly during the winter and spring, the disease is so common that the number of cases reminds us of an epidemic. In summer and autumn the cases are usually solitary.

Anatomical Appearances.—We do not often have the opportunity of examining the bodies of persons who have died during an attack of acute articular rheumatism. When we do, the changes found in the joints are often unimportant, being limited to a moderate hyperæmia of the synovial capsule and a slight increase and turbidity of the synovia. In some cases even these do not remain to tell of the inflammation that has undoubtedly existed during life, and the result of the autopsy is almost as negative as in most cases of erysipelatous dermatitis. On the other hand, on *post-mortem* examination, we occasionally find the synovial capsule of some joints of a dark-red color, from hyperæmia and ecchymosis, relaxed and puffed up, the joint dilated and filled with purulent fluid; the ends of the neighboring bones even may be injected and contain extravasations of blood. In all recent cases the blood contained in the heart and large blood-vessels shows very plentiful deposits of fibrin. There are also the frequent anatomical changes due to the complications of articular rheumatism, particularly of pericarditis, endocarditis, and myocarditis.

Symptoms and Course.—In some cases the outbreak of the disease is preceded for a few days by general uneasiness and a feeling of weariness in the limbs. In others, the premonitory symptoms are wanting, and the attack begins suddenly and unexpectedly. The onset of the disease is not frequently marked by a chill, as we have learned is the case in pneumonia and some other inflammatory diseases. Usually there is only a slight rigor, which is often repeated during the first few days; in the other cases even this is absent, and there is from the first the hot feeling, which otherwise does not occur till after the chill. Just as the symptoms of fever begin, or soon afterward, the patients complain of pain in one or usually in several joints; this is moderate at first, but quickly and steadily becomes more severe

and soon grows very decided. As long as the patient makes no attempt to move the affected joint, and it is not touched, the pain is endurable; but every attempt to move the joint or the slightest pressure on it, in severe cases even the weight of the bedclothes, increases the pain so that the patients often moan and cry till they have remained quietly for a time in a comfortable position protected from any injury. If we examine the affected joint, we sometimes find it moderately, sometimes very much swollen. As we have already said, the swelling only partly depends on the effusion into the joint; being partly due to œdema of the skin and subcutaneous tissue, it usually extends to the neighboring parts of the limb, and on careful examination it looks as if the ends of the bone were enlarged. The skin over the affected joint is either of normal color or else light, or exceptionally dark red. The degree of the swelling and the severity of the pain are not always in exact proportion; the pain is often very severe, while the swelling is scarcely observable. The large joints, particularly the knee, foot, hand, elbow, and shoulder, are most frequently affected; but the small joints, especially the finger, sterno-clavicular and intervertebral articulations, are be no means exempt. The toe-joints are only exceptionally implicated. Occasionally also the symphisis pubis is attacked. Not very unfrequently the inflammation extends to the muscles and fascia about the affected joint. The number of joints involved varies. Even at the commencement of the disease it is rare for a single joint to be attacked; far more frequently, two, three, or even more, are simultaneously diseased. It is customary, as the disease progresses, for joints, that have previously remained free, to become involved, and for the symptoms to reach their height in these, while it is subsiding or has even disappeared in those first inflamed. If the pain and swelling disappear slowly in the joints first seized, while they develop in others, the number of joints implicated at one time may be very large, and the patient's state be very helpless and pitiable. Occasionally such patients cannot make any movements; they are unable to stir from any position in which they have been placed; they fear even the most careful passive motion rendered necessary for urination, defecation, eating, or drinking. Even slightly shaking the bed increases their pain, and induces groans and complaints. But this extent and severity of the symptoms are not frequent. In most cases two or three, or at least only a moderate number of joints, are simultaneously affected with severe pain, while the others remain free, or are somewhat stiff and only pain when freely moved; sometimes also they crepitate.

Fever usually accompanies the commencement of the disease; occasionally it precedes the local symptoms, and only exceptionally

comes on late; it usually corresponds to the severity and extent of the local symptoms, and has all the characters of a sthenic inflammatory fever. The bodily temperature does not usually attain the high grade observed in infectious diseases; in most cases it does not rise more than two or three degrees above normal, but exceptionally it becomes 104°—105.5°, or even higher. The pulse is more frequent, usually from 90—100 beats in the minute, and is generally full and soft. Only in those cases where the temperature becomes very high does the pulse reach 120—130 beats in the minute. In many cases, even where there is no complication with endocarditis, we hear blowing murmurs, so-called blood-murmurs. The respiration is hastened, the skin is almost always covered with very profuse perspiration of acid odor, which has no critical significance, but is usually just as abundant during the advancing stage, and at the height of the disease, as in the declining stage. The skin is often covered with an eruption of miliaria rubra. We have previously shown that this exanthema belongs to the eczemas, and results from irritation of the skin at the orifices of the perspiratory glands from excessive sweating. Besides vesicles filled with a milky fluid, we often see efflorescences in the shape of red papules, where there has not been sufficient exudation to raise the epidermis into a vesicle. More rarely sudamina, or miliaria alba, occur. The great loss of water, due partly to increased evaporation caused by the high temperature, partly to the profuse sweating, is shown not only by the thirst, but by the scantiness of the urine. Frequently only twelve to fourteen ounces of urine are evacuated in twenty-four hours. As the amount of urea is increased on account of the accelerated change of tissue, the concentrated urine has a very high specific gravity; and, as the amount of water it contains is insufficient to retain the urates in solution at low temperature, copious sediments of these salts form as soon as the urine cools. These appear darker red than common, on account of the quantity of coloring matter which is deposited with the salts. A very copious sediment of urates does not always allow us to conclude that the amount of urea excreted in twenty-four hours is increased; at least, in two cases of acute articular rheumatism, treated at the Greifswalder clinic, where *Hoppe-Seyler* had the kindness to examine the very thick urine which deposited a large sediment, the amount of uric acid was only normal. It is, to some extent, justifiable to term such urine almost pathognomonic of acute articular rheumatism; for in scarcely any disease is the loss of water so great from profuse sweating as well as from increased evaporation, caused by the high temperature.

Among the complications of acute articular rheumatism, pericarditis, endocarditis, and myocarditis, deserve especial mention. When

speaking of heart-diseases, we fully showed that the frequency of these complications has been greatly exaggerated, particularly by *Bouillaud.* Apparently, *Bouillaud* considered inflammation as the cause of the above-mentioned "blood-murmurs," which are probably due to an abnormal tension and consequent irregular vibration of the valves and walls of the vessels. We have mentioned that, according to *Bamberger's* statistics, the complication with endocarditis occurs in about twenty per cent., with pericarditis in about fourteen per cent., of the cases of acute articular rheumatism, while myocarditis is much rarer. According to *Bamberger's* observation, the tendency to complicating inflammation of the heart and pericardium is greater in proportion to the number of joints affected. In these cases the heart-affection is a complication and not a metastasis, as is shown by the fact that, when any form of carditis develops, the affection of the joint continues as it was previously. For the symptoms and course of the heart-affection, we will also refer to what has been said, and shall only again call attention to the fact that, in many cases, there are no subjective symptoms, and the complication is only recognized by physical examination Pleurisy and pneumonia are far rarer complications than the different forms of carditis, and still rarer ones are cerebral and spinal meningitis. In protracted cases, suppuration of the joints may lead to pyæmia; but, as this termination of idiopathic arthritis is rare, such cases are very uncommon.

The disease has no cyclical course, and lasts, with varying intensity, in mild cases, one or two weeks; in severe ones, for many weeks. The pain and fever usually remit in the morning, and exacerbate in the evening; but these variations are not regular. Frequently, cases, which at first appeared mild and favorable, subsequently become very obstinate and malignant; and, in the midst of an apparent improvement, which has given hopes of a speedy recovery, the fever and local symptoms suddenly become worse again, and advance to a height not previously attained.

In regard to the results of acute articular rheumatism, we must especially bear in mind that not only the mild cases, where few joints are affected and the fever is slight, may recover completely, but that recovery is also the usual termination for severe cases, where numerous joints are inflamed, very painful, and much swollen, and where the severe local symptoms are accompanied by corresponding fever. But, even in favorable cases, recovery does not take place with sudden subsidence of the symptoms, but with their gradual and not always regular diminution. Notwithstanding its treacherous course and its frequent complication with inflammation of the most important organs, this malady rarely terminates fatally. Death seldom occurs, except when

the cardiac complications are unusually severe and are accompanied by inflammation of the lungs and pleura; even in such cases, recovery often takes place, contrary to all expectation. Occasionally, also, death occurs with the symptoms of sudden collapse, when there is no complication, being preceded for a short time by delirium, coma, or other symptoms of great nervous disturbance. On autopsy, in such cases, no changes can generally be found in the central organs of the nervous system; hence it has been supposed that they depend on an unexplained blood-poisoning. But it seems to me that this hypothesis is unjustifiable, till careful observations of the bodily temperature have shown that, in these cases, the fatal result is not due to the fever and the increase of the temperature to a point fatal to life; particularly since it has been shown that, in this disease, the temperature, which is usually moderate, may rise quite high. In a few instances, moreover, the severe brain-symptoms depend on inflammation of the meninges, that is, on changes analogous to those in the joints. I myself have only seen one case where the moderate increase of bodily temperature, the delirium in the first stage, and the subsequent coma, together with excessive retardation of the pulse and repeated vomiting, left no doubt as to the nature of the disease. This case ended in recovery, so that the diagnosis was not confirmed by autopsy; but in a dissertation written by Dr. *Flamm*, under the direction of my colleague *Köhler*, on "Meningeal Symptoms in Acute Rheumatism," cases are reported where *post-mortem* examination showed the presence of inflammatory disease of the meninges. It has not yet been decided whether the chronic cerebral disturbances, occasionally left after acute articular rheumatism are due to perceptible anatomical changes. *Griesinger* speaks of them as follows: "The severe cerebral disturbance, lasting for months or more, caused by articular rheumatism, appears as insanity without fever, characterized by depression, or as pronounced melancholy with stupor. It may be followed, or may alternate, with states of excitement; sometimes this disturbance is accompanied by convulsive choreic movements; the prognosis is, on the whole, favorable; recovery seems to result most rapidly and certainly where, after a time, during the cerebral disturbance, the joints are again attacked with acute rheumatism." Acute articular rheumatism very often ends in incomplete cure. In many cases the fever, severe pain, and the swelling of most of the joints disappear, but a chronic rheumatism remains in certain joints, proves very obstinate, and often never entirely disappears. Still more frequently the patients live through the acute articular rheumatism and its complications, but acquire valvular disease of the heart, which is never cured, and of which they soon die. After the disease has lasted a long time, particularly if the fever was severe,

the convalescents are usually very anæmic, and we may hear false murmurs in the heart and great vessels, which, on careless examination and insufficient attention to the general state of the patient, may be confounded with valvular affections.

Treatment.—For acute articular rheumatism all kinds of antiphlogistic remedies have been used, particularly general and local bleeding, nitrates of potash and soda, tartrate of antimony in small doses, and many have thought it possible to shorten the course of the disease, and to prevent the complications, by energetic antiphlogistic treatment. From what we have said of the varying course and duration of the disease, it is evident that it would be very difficult to prove any such suppositions. On the other hand, if energetic antiphlogosis did have a beneficial effect in some cases, the fact that the disease has often persisted for weeks and that heart-affections have often developed in spite of it, speaks against its infallibility. Hence, in recent times, the treatment by repeated bleeding, by large, and even dangerous doses of saltpetre, as well as the systematic use of tartar emetic, has been abandoned, and we now employ other remedies which may, perhaps, be as uncertain, but are, at all events, less dangerous. Nitrate of soda, in moderate doses (℥ ij to ℥ vi of water, a tablespoonful every two hours), has alone maintained its position in the treatment of acute articular rheumatism, and is prescribed by most physicians, without any great expectations from it, just as it is given in pneumonia and other febrile diseases. Since this remedy can hardly prove injurious in small doses, and since its antiphlogistic action has not been disproved, and is not at all improbable, and, finally, as we know no other remedy that will certainly arrest or shorten the course of acute articular rheumatism, we have nothing to say against its employment. Perhaps it has been wrong to follow *Rademacher's* advice, and prescribe nitrate of soda instead of nitrate of potash, which is probably more active. Recently, great authorities recommend a return from cubic nitre to common nitre (*Köhler*). The next most popular remedies are colchicum (tinct. or vin. sem. colchici ℥ ss, tinct. opii ℥ ss, 15—20 drops every three hours), lemon-juice (℥ ss three times daily), iodide of potassium (℥ ss— ℥ j dissolved in 3—4 oz. water, during the day), quinine and the narcotics, especially opium and morphine We cannot, with our present experience, give any special indications for the employment of colchicum, lemon-juice, and iodide of potassium, which seem to have proved serviceable in some cases, but most generally have no effect on the course of the disease. In very obstinate cases, with frequent relapses, we will employ these remedies, slight as is the hope of benefit from them. Quinine is no more a specific in acute articular rheumatism than in pneumonia, typhus, and many other dis

eases in which it is, nevertheless, an important remedy. But it is one of the most powerful antipyretics, and it should be used when the fever is high. It is customary to give a scruple or half a drachm during the day, and it is best to give such large doses, for, according to my experience, it is only from large doses that we can expect any decided effect on the bodily temperature and frequency of the pulse. After the experiments of *Weber* and *Billroth*, there can scarcely be any doubt that the quality of the blood in fever patients decidedly favors inflammatory disturbances of nutrition. If this be so, an antipyretic treatment has also an antiphlogistic action, and quinine as well as other antipyretic remedies would answer not only the symptomatic indications, but also the indications from the disease, particularly in acute articular rheumatism, where, while the fever continues, new joints are continually becoming affected. At all events, quinine deserves a full trial in acute rheumatism, when there is any great increase of bodily temperature. Most of the narcotics can be dispensed with; but opium and morphine are not only invaluable remedies for moderating the sufferings of the patient, but it also seems as if their exhibition prevented the inflammation in the joint reaching so high a point. I shall not attempt to say whether they shorten the course of the disease, as some observers claim they do; but I can affirm that, even where there is great fever, large doses of opium or morphine are well borne. Where the pain disturbs the night's rest, it is advisable to give the patient a grain of opium, or one-quarter of a grain of morphia, in the evening; and, when the pain is uncommonly severe, we may order this dose every two hours, till relief is obtained. In the treatment of most cases of articular rheumatism, I limit myself to the exhibition of quinine and opium. Among the *external remedies*, warm and cold compresses, leeches, blisters, as well as numerous narcotic and irritant lotions and liniments are recommended. As might be supposed from their number, these remedies have, on repeated trials, been of less service than was claimed by the person proposing them; we must acknowledge that external remedies also usually have no effect on the course of the disease, and are at most only palliatives. Any physician in good practice will not deny that cases of acute articular rheumatism, coming under treatment at the commencement of the disease, often resist all external or internal treatment for three to six weeks or more.

In cases of moderate intensity, it is well to envelop the affected joint in wadding; where the pain is severe, cool evaporating lotions are best, particularly as ice and cold-water compresses are popularly considered as bad antirheumatics and are consequently used irregularly. Elaylchlorüre (3 ss--3 j, lightly rubbed in), as recommended by *Wun*

Aerlich, is an excellent palliative; but frictions with ether, which I used on economical grounds as a substitute for elaylchlorüre, answered the same purpose. Local abstractions of blood should be limited to those cases where a single joint has been considerably swollen and painful for a long while. If, after repeated leeching, the pain and swelling do not subside, if they remain fixed in one joint after disappearing from the rest, we may cover it with blisters, or paint the skin with tincture of iodine. When ordering the diet of the patient, we must bear in mind the exhaustion threatened by the fever. Hot drinks should be forbidden, as they uselessly increase the perspiration. The room should not be kept too warm, but its temperature should be regular. The complications are to be treated as advised elsewhere. If, during the disease, severe cerebral symptoms come on, we must decide whether they depend on excessive increase of the bodily temperature, or on inflammation of the meninges. In the former case it will be well to reduce the temperature by wrapping the body in wet sheets, or by cool baths. Both should be repeated as often as the temperature threatens to increase to a great height. If, when the brain symptoms occur, the temperature be not greatly increased, we should apply leeches to the head, and then cover it with frozen compresses.[2]

CHAPTER II.

CHRONIC ARTICULAR RHEUMATISM—RHEUMATISMUS ARTICULORUM CHRONICUS—RHEUMARTHRITIS CHRONICA.

Etiology.—Chronic articular rheumatism is the name applied to a chronic idiopathic inflammation of the joints, which usually attacks only one or a very few joints, which passes from one joint to another far more rarely than acute articular rheumatism does, and which, in spite of its long duration, induces comparatively little anatomical change. When chronic articular rheumatism leads to suppuration of the joint and caries of the ends of the bones, it is generally customary not to consider it any more as chronic articular rheumatism, but as chronic inflammation of the joint, and to transfer it to surgery just as we do arthrocace and tumor albus. Chronic deforming rheumatism we shall treat of in the next chapter. Chronic articular rheumatism often develops from the acute thus: after the disease has run its course in the other joints, one or more of them do not return to the normal state, but remain the seat of permanent disturbances. In other cases it appears as a chronic disease from the start. Very frequently there is a decided congenital predisposition to chronic articular rheumatism; on

the other hand there are numerous cases where such a predisposition is acquired. First among the predisposing causes are previous attacks of acute articular rheumatism, for after these there often remains a peculiar tendency to the chronic form which did not previously exist. The same is true of one or more attacks of chronic articular rheumatism, as this also usually leaves behind a so-called rheumatic predisposition. The most frequent exciting cause is catching cold, but especially a protracted sojourn in cold, damp, and windy places. Thus almost all old wash-women suffer from chronic articular rheumatism. In many cases the exciting cause cannot be determined.

Anatomical Appearances.—In the comparatively rare cases where we have an opportunity to examine joints that have been the seat of chronic articular rheumatism, we generally find the synovial capsule and the ligaments of the joints thickened, the fringe-like processes of the membrane hypertrophied, and not unfrequently degenerated, the cartilages relaxed and shaggy, the synovia is cloudy.

Symptoms and Course.—Chronic articular rheumatism appears under two different forms. In the first form, single joints are often for months or years the seat of constant pain. This is increased by pressure, but particularly by active or passive motion, and there are also severe paroxysms of pain which apparently come on spontaneously, especially at night. If we place the hand on the joint in motion, we often perceive a distinct crackling or crepitation. Occasionally the joints are decidedly swollen; but the swelling does not depend on inflammatory œdema of the subcutaneous tissue, as is the case in acute articular rheumatism, but entirely on an increase of the synovia in the joints, and on thickening of the capsule and ligaments. In other cases there is no swelling, as there is no considerable effusion in the joint, or else the swelling is only apparent; the joint becomes more prominent, because the muscles are atrophied on the affected limb, which is always less used. Where this form lasts a long time, incomplete, false anchyloses are readily developed; but it rarely leads to tumor albus, or arthrocace. The second form of chronic articular rheumatism consists mainly of a series of attacks of acute articular rheumatism, occurring at short intervals, in which certain joints are always affected. Persons suffering from this form often become "rheumatic" at every change of the weather, every time they are exposed to a draught, and just as often without any perceptible cause. Sometimes one joint is affected, sometimes another; it is slightly swollen, very sensitive to pressure, but particularly painful when moved. The fever, which is almost always present, is evinced by continued frequence of pulse, constant perspiration, by thick sedimentary urine, as well as by the gradually increasing apathy and emaciation

of the patient. This form also of chronic articular rheumatism is very obstinate, and, once rooted, often lasts for life. It is frequently complicated with muscular rheumatism, and not unfrequently with those forms of neuralgia and paralysis which are usually termed rheumatic.

TREATMENT.—The form of chronic articular rheumatism, limited to certain joints, requires chiefly a local treatment; while vague chronic articular rheumatism, on the other hand, requires to be treated generally. In recent cases, the best local treatment is by leeches or wet cups. To attain satisfactory results, these should be frequently repeated at moderate intervals; they then do excellent service, and cannot be replaced by any other remedy. Even in obstinate cases it is well to begin the treatment with leeches or wet cups, unless contraindicated by the general state of the patient. The result of the first abstraction of blood must decide us whether to repeat it in such cases also, or to use derivatives instead. Among the latter, such as redden the skin, or cause superficial inflammation of it, are to be preferred to those which, from their volatility, irritate the nasal mucous membrane, but cause no perceptible change in the skin. Heretical as it may sound, we still believe, that when the disagreeable opodeldoc, volatile liniment, spirits of camphor, as well as the fragrant mixtura oleoso-balsamica, prove beneficial, their efficacy is chiefly due to the manipulation of the parts. For support of this assertion, we may refer to the fact that of late even the laity, who formerly used the above remedies as a matter of course in all rheumatic affections, use them less frequently, and employ frictions with French brandy and salt instead. The application of sinapisms and frictions with spirits of mustard, by which the skin is temporarily reddened, are sometimes of undoubted benefit, and, when continued regularly, may cause permanent improvement. Frictions with veratrine and chloroform liniment induce peculiar sensations in the skin, which, to some extent, speak for their derivative action. In mild cases I have employed a solution of veratria (gr. vj—x), in chloroform (℥ ss), and mixt. oleoso-balsamica (℥ ij), with apparently favorable effect. Vesicants are far more efficacious than rubefacients; among these may be classed painting the skin with tincture of iodine, as it causes the epidermis to be thrown off; and, if the undiluted tincture be used, it often induces blisters. In very obstinate cases, in order to secure a good result, the portions of surface denuded of cuticle must be kept suppurating for a time. I consider a powerful douche over the affected joint as the most efficient derivative, and even place it before the hot iron and moxæ, which are also advised in localized articular rheumatism. We may readily satisfy ourselves that an energetic douche will leave a hyperæmia of the skin lasting for several hours I have no personal experience of the

action of the steam douche. Besides the above-named local remedies, and the popular resin and narcotic plasters, the application of "gout-paper" (gicht papier), skins of animals, wadding, raw wool, cotton, etc., which partly favor derivation to the skin, partly maintain an equable temperature, other remedies are also used locally, which are expected to be absorbed, and consequently act favorably on the course of rheumatism. Chief among these remedies are iodine and mercury. Little as I recommend frictions with mercurial ointment in these affections generally, I must still decidedly advise continued frictions of ointment of iodide of potassium, or of mercurial ointment, with iodide of potassium (℈j—℥ ss), in old cases of fixed arterial rheumatism, where abstraction of blood and derivatives have proved inefficacious. The second of these salves causes slight derivation to the skin at once, and, after a few days, there is an eczematous eruption of the part. Painting the part once or twice daily with a *Lugol's* solution (iodine ℈ss, potass. iod. ʒ ss, aq. dest. ℥ j—ij) does excellent service. Among the most efficient methods of treating chronic articular rheumatism is the constant current of electricity. My experience with it is not very great, it is true; but it fully confirms the reports of *Remak* and *Erb*, that even obstinate cases of chronic articular rheumatism are usually cured after a few sittings. I pass quite strong currents through the affected joint, by applying thin metallic plates to the skin over the joint, instead of using the common olive-shaped electrodes. When the pain becomes too severe at the point where the negative pole is applied, I change the electrodes. The very favorable results of the constant current in chronic articular rheumatism confirm me in the belief that its results in neuralgias and paralysis also depend on its catalytic action. In the constitutional treatment, which must take a prominent part in the vague form of chronic rheumatism, and must be used in connection with local treatment in the fixed form, the systematic employment of warm baths deserves most confidence. As no physician of experience will deny, many persons are perfectly cured by one or more courses of treatment at Wildbad, Gastein, Pfäffers, Ragaz, Teplitz, Wiesbaden, Rehme, etc., who had previously resisted all kinds of treatment for years. The fact that warm springs of very varied chemical composition, as well as those whose waters contain very little mineral matter, have equal reputation in chronic rheumatism, indicates very decidedly that the effect is much more dependent on the bathing in warm water than on the different qualities of the water. In well-constructed hospitals, provided with suitable baths, just as good results are attained in the treatment of chronic rheumatism as at Aix-la-Chapelle, Teplitz, and Wildbad, as I can show by some very striking although not very numerous cases from the Greifswalder clinic. In

private practice it is difficult to conduct the bath-treatment with the necessary care and prudence; hence it is generally better to send patients, whose means permit, to one of the above-mentioned warm springs. Baths of 95°—100° are sufficiently warm, and are probably to be preferred to those of higher temperature; at least, according to my experience, Russian vapor-baths do less good in chronic rheumatism than warm-water baths. The same observation has been made elsewhere. The erection of a Russian vapor-bath, at a place where there has formerly been none, usually pays very well the first and second year, for most persons with chronic rheumatism crowd to it, expecting to be cured; but the third and fourth years the number of customers is usually much less, and this is certainly not because the persons with rheumatism have all been cured, but because their hopes have been disappointed. From the experience at Wildbad, Leuk, and elsewhere, it seems advisable to gradually extend the duration of the bath, and, toward the end of the treatment, to let the patient pass an hour or more in it. It is very important for them to avoid taking cold after bathing, and it would be well to give them a blanket-sweat. It is customary to give about thirty warm baths in succession, and if this number proves insufficient, it seems better to stop their use, and take up the treatment again a few months later. In "cold-water cures," patients with chronic rheumatism, especially old cases, are not usually cured. I know not a few cases of this class who left the cold-water cure establishment, after a sojourn of several months, sicker than when they entered. In recent cases, on the contrary, hydropathy appears to have a favorable effect. Among the "anti-rheumatic" medicaments, tinc. colchici sem. (℥ ss), with extract. aconit. (ʒ ss), and tinc. opii (ʒ ss), in doses of 15—20 drops four times daily, has a great reputation, and is probably the most common prescription in chronic articular rheumatism. Unfortunately, we do not yet know in which cases colchicum (which is certainly not altogether inactive) is indicated, nor when we cannot expect any thing from it. Ammoniated tincture of guaiac. and the antimonials, which formerly had the reputation of being active anti-rheumatics, are little used of late. In some cases of chronic articular rheumatism, and, as it seems, chiefly in those cases where the ligaments are mostly affected, iodide of potash in large doses (ʒ j— ʒ j daily) appears to be very serviceable. Improvement ordinarily begins with the appearance of slight iodism, and it is advisable to increase the dose until the effect of the medicine is observed on the skin, or the mucous membrane of the nose. In recent cases, and in young persons, the regimen of the patient must differ from that in old cases, or patients of advanced age. While, in the former, especially after improvement has begun, we should advise a careful

hardening process by washing in cold water, cold bathing in river or sea, regular walks in all kinds of weather, etc., in the latter, all opportunities of catching cold should be avoided, the sea and river baths forbidden, and the patient should be advised to wear flannel next the skin. If the sleeping-room be dark and damp, we cannot too strongly urge its exchange for a healthier and drier one.

CHAPTER III.

DEFORMING ARTICULAR INFLAMMATION, ARTHRITIS DEFORMANS, ARTHRITIS NODOSA, ARTHRITIS PAUPERUM, ARTHRITE CHRONIQUE SÈCHE.

Etiology.—By arthritis deformans, we mean those forms of articular inflammation where not only the synovial capsule and ligaments of the joint exhibit the signs of a chronic inflammation having no tendency to suppuration, but where, at the same time, the cartilages and surfaces of bone in the joint show peculiar changes characteristic of this form of arthrophlogosis. The latter consist chiefly of a loss of the articular cartilage and surface of the bones, and in a central induration of the epiphyses, accompanied by a proliferation of bone-substance at the periphery. Arthritis deformans is by some authors regarded as a peculiar form of chronic articular rheumatism, while others consider it as essentially different from the rheumatic affections. From the great elasticity of the term rheumatism, one view is as correct as the other. Arthritis deformans has at least this in common with the rheumatic inflammation of the joints described in the last chapter; it cannot always be traced to catching cold from living in damp places exposed to draughts. Arthritis deformans never occurs during childhood. Solitary cases are seen about the age of puberty; it is most frequent between the twentieth and fortieth years; but it also occurs later in life, and even in advanced age. It attacks women more frequently than men. It is so much more common among poor people than among the wealthy, that it has been termed arthritis pauperum. We do not know whether this depends on the bad, damp dwellings, on the want of good nourishment, or on some other causes to which the poor are more exposed than are the well-to-do classes.

Anatomical Appearances.—On anatomical examination of the swollen, misshapen joints, we find the articular capsule decidedly thickened and covered with ragged proliferations. The joint contains a very small amount of synovia (hence the name *arthrite chronique sèche*). The articular cartilages are broken down into filaments, occasionally ossified, and in advanced cases they have entirely disappeared

by wasting away, so that the ends of the bones come in contact, and have smooth, articular surfaces. As a result of inflammatory atrophy the central parts of the epiphyses appear porous, while their size is decidedly increased, either regularly or in warty protuberances, by the formation of osteophytes.

Symptoms and Course.—Arthritis deformans results from acute articular rheumatism much less frequently than the above-described chronic inflammation does; in most cases it develops slowly, and gradually reaches its height. A halt sometimes occurs in its progress, but never a retrogression or a disappearance of the existing deformity. At the commencement of the disease the patients complain of pain in the affected joints; this is usually slight, but is sometimes so severe as to rob them of sleep. The pain is increased by pressure, and still more by movement of the joint; if the hand be laid on it, we may almost always perceive a crackling or crepitation. The affected joints seem decidedly thicker; the skin covering them is of normal color, or slightly reddened. The above changes most frequently affect the joints of the hand and feet, especially those of the fingers, toes, and the metacarpal and metatarsal articulations, but they may also attack any of the other joints of the body and even the spinal column. Cases where the patient is confined to his bed for years, or sits up during the day in an arm-chair and at night is carried to bed, may be found in almost any large poor-house. The laity usually call this state being "drawn up by the gout." The symmetrical appearance and progress of the disease on the two sides of the body are often very remarkable. It is rare for one hand to be affected first, and the other one subsequently; the process is apt to begin simultaneously in both hands, and to pass to the two feet at the same time. Characteristic subluxations almost always occur in the affected joints, especially in the fingers; these are hardly explained by the widening of the epiphyses over which the tendons pass, by the destruction of the articular cartilages, and the smoothing off of the bony surfaces of the articulation. The phalanges are almost always flexed on the metacarpal bones, and are at the same time deviated toward the ulnar sides, so that the fingers lie over each other like the shingles in a roof. In the phalanges themselves the subluxations are sometimes in the flexed, sometimes in the extended position, in the different joints. Patients with this tedious disease may attain very old age. It is remarkable that, in spite of the long duration, there is rarely complete anchylosis. The above process sometimes occurs exclusively in the hip-joint, and then either induces a wearing off of the head of the bone, or the formation of large stalactite-like osteophytes, about its periphery. This disease, called *malum coxæ senile*, belongs to surgery.

Treatment.—Little hope as we have of removing by treatment the deformity that has once developed, I cannot agree with those authors who say that treatment is entirely useless in arthritis deformans. The impairment of motion does not usually depend solely on the deformity, but to a considerable extent also on the still existing inflammation. The remedies which we placed in the first rank against chronic articular rheumatism, particularly the warm baths and the painting with tincture of iodine, have in many cases at least a favorable effect on the inflammation. Every season at Töplitz, as I actually know, and probably also in the other watering-places mentioned in the preceding chapter, some patients with arthritis deformans have improved so much that they were able to leave the chair with rollers, and after the termination of their treatment could roll other patients to the baths.

CHAPTER IV.

MUSCULAR RHEUMATISM—RHEUMATISMUS MUSCULARIS.

Etiology.—Under the term muscular rheumatism it is customary to class not only rheumatic affections of the muscles, but also those of the fascia, periosteum, and other fibrous tissues, except those about the joints. The changes induced in these tissues by rheumatic affections are not exactly known. The negative results of most autopsies seem to render it very probable that the anatomical changes are of that class that leave little trace in the cadaver, that is, that they consist chiefly in hyperæmia and scanty serous exudations. In rare cases, however, the rheumatic process appears not to be arrested at this point, but to lead to inflammatory proliferations of connective tissue. *Froriep* and *Virchow* found places in the muscles where the muscular tissue was replaced by callous connective tissue (rheumatic callosities), and *Vogel* observed thickenings and adhesions of the neurilemma of the corresponding nerves in several cases of chronic rheumatism. These cases are so rare compared with those where no changes can be discovered, that it is hazardous to base a definition of rheumatic affections on them. Even supposing that hyperæmia and serous exudations, and in severe cases an inflammatory proliferation of connective tissue, be the basis of the rheumatic affection, it still remains undetermined whether the sensory nerves traversing the muscles are morbidly excited by changes in the muscles and sarcolemma, or by simultaneous changes in their neurilemma. In the latter case, muscular rheumatism would be a so-called rheumatic neuralgia of the small nerves supplying the muscles. Regarding the etiology of muscular rheumatism, we

may refer to what we have said of the etiology of acute and chronic articular rheumatism, and the more so as muscular rheumatism is very often complicated with the articular form of the disease. Catching cold is certainly the most frequent cause; but the sudden occurrence of some forms, as lumbago, renders it very probable that, besides catching cold, there are other causes, especially straining or fatigue of the muscles, etc.

Symptoms and Course.—The most important, and usually the only symptom of muscular rheumatism, is the pain which is generally designated as stretching or tearing. Moving or rubbing the affected tissues increases this pain, while regular pressure generally diminishes it. Occasionally the pain is accompanied by inability to shorten the affected muscle, and to make active movements with it. The skin covering the part does not appear red and swollen, or warmer than the surrounding parts. The pain usually exacerbates toward evening, and remits in the morning. It is generally made worse by cold and dampness, and improved by dry warmth. But sometimes the warmth of the bed increases rheumatic pains. Occasionally muscular rheumatism is "wandering," that is, the pain leaves one place and appears in another. Sometimes it remains "fixed" in certain muscles, fascias, etc. In most cases muscular rheumatism is an acute disease, which entirely disappears after a short duration; rarely it is a chronic affection, and either the wandering or fixed form may become chronic, just as wandering or fixed articular rheumatism does. According to its location, muscular rheumatism has received various and sometimes peculiar names. If the frontal, occipital, or temporal muscles, the galea aponeurotica, or the periosteum of the skull be affected, we term it a *cephalalgia rheumatica*. We should make it a rule not to misuse this name, and in each case, before making a diagnosis of cephalalgia rheumatica, to determine carefully if the above tissues be really the seat of the pain, whether movement of their fibres or contraction of the muscles increases the pain, and, finally, whether the affection be primary and idiopathic. Rheumatism of the head is most readily mistaken for neuralgia, or syphilitic periostitis. It is a very common, bad habit, to ascribe to rheumatism the numerous cases of severe and obstinate headache whose true cause we cannot discover. The laity, who do not make any great distinction between rheumatism and gout, generally call all obstinate headaches gout in the head. If the muscles of the neck be affected with rheumatism, the movements of the head become painful; the patients fear and shun them. There is "stiff neck," an affection which complicates many cases of angina faucium. If the muscles of only one side of the neck be affected, the head is held constantly to one side ("wry neck," *torticollis rheumaticus*). This affec-

tion, which is generally mild, and subsides in a few days, becomes chronic, and may induce permanent shortening of the muscles in some cases, which, however can hardly be distinguished from the previously described spastic form of torticollis. Rheumatismus pectoris, or *pleurodynia rheumatica*, occurs chiefly in the pectoralis major and intercostal muscles. In the former case, the movement of the arm forward and motion of the affected muscular filaments are painful; in the second case, the respiratory movements, and especially coughing and sneezing, as well as bending the thorax laterally, are so. From the hinderance to the respiration, these patients look as if the pain were deeper seated—as if the lung or pleura were diseased; and as moving the skin, fascia superficialis and pectoralis, does not increase the pain, the physician also might be deceived, if moving the intercostal muscles from before backward with the point of the finger did not render the pain unbearable, while laying the hand on the part and making even pressure usually moderates it; and if the absence of cough, and especially the physical examination of the breast, did not confirm the diagnosis. Rheumatism of the back, particularly of the shoulders, *omodynia rheumatica*, is among the most frequent forms of muscular rheumatism; it is easily recognized by the impaired motion of the shoulder-blade and arm, as well as by the severe pain induced by moving the filaments of the trapezius, latissimus dorsi, and deltoid, or if the deeper layer of muscles be affected by the stiff position of the patient, and the severe pain induced by stooping. Painful affections of the abdominal muscles occur chiefly after severe straining in coughing, and, from their severity, may excite the suspicion of severe disease. Noting the state of the pain on regular pressure over the abdomen, and on movement of the muscular filaments, is the best means of avoiding error. Rheumatism of the lumbar muscles and lumbo-dorsal fascia, *lumbago rheumatica*, is remarkable for its severity and its frequently very rapid occurrence. Not unfrequently, patients who, a few minutes previously could move with perfect ease, cannot rise from their seat, or at least suffer severely while doing so. When they go to bed, or get up also, or even when they wish to sit up in bed, or on any attempt to move the lower part of the spinal column, they make wry faces, cry out, and place themselves in the most peculiar attitudes to favor the painful parts, and attain their end without motion of these parts. Hence, patients suffering this "witch's spell" ("Hexenschuss") excite laughter as well as pity. Lastly, all the muscles of the extremities may be attacked with rheumatism either individually or in groups, so that sometimes one and sometimes another set of motions becomes painful or impossible.

Treatment.—The same principles answer for the treatment of

muscular rheumatism that we laid down for that of chronic articular rheumatism; that is, in the fixed form the treatment must be rather local; in the wandering form, more general. But, as the former is usually a milder and less obstinate disease than the latter, less energetic treatment is necessary in most cases. The most successful local treatment is abstraction of blood, by wet cups, if possible. The life-invigorator (Lebenswecker) has, of course, just the same effect as cupping; formerly its effect in rheumatism was regarded, even by physicians, as miraculous. The success of irritating liniments, blisters, sinapisms, plasters, gout-paper, or skins of animals, in muscular rheumatism, is about the same as in articular rheumatism. Perhaps one of the most effective, but at the same time most painful, rubefacients, is the application of the induced current by means of the electrical brush. The same is true of the use of the constant current in this affection as in chronic articular rheumatism. I can fully confirm the brilliant results reported by *Erb*. Stroking and kneading the painful muscles, which forms the regular business of some persons called "rubbers," is one of the most efficient means of local treatment. Diaphoresis is the most reliable constitutional remedy. Treatment by baths is rarely necessary, and is so only in obstinate cases which are usually complicated with chronic articular rheumatism. A few cups of elder-blow tea, or some warm drink, and plenty of blankets over the patient to excite profuse perspiration once or twice, usually improve or remove muscular rheumatism. In recent cases a vapor-bath often gives instant relief; and although I have spoken against the continued use of vapor-baths in chronic articular rheumatism, I can recommend the trial of one or two in recent cases of muscular rheumatism.

CHAPTER V.

GOUT—PODAGRA—ARTHRITIS.

Etiology.—There is scarcely any disease that shows so great a similarity of symptoms at the commencement of all cases, and which consequently is so markedly a peculiar disease, as gout. It is true, old cases may resemble other diseases, particularly rheumatism; but, in such cases, it is only necessary to inquire about the commencement of the affection, in order to prevent its being mistaken for rheumatism. But although it is certain that gout is a peculiar form of disease, and although constant chemical changes have been observed in the blood of gouty patients (*Garrod*), and uric acid, the substance which is in excess in the blood, has been found in the products of gouty inflammation, still we have no exact knowledge of the pathogeny of this

disease. We neither know whether the "uric-acid diathesis" be the primary and chief anomaly in gout, and whether it be not accompanied by other and more important changes in the composition of the blood; nor do we know the disturbances of nutrition by which one of the constant products of normal nutrition, uric acid, is formed in excess. The remote causes of gout are somewhat better known. It is proved by statistics that *hereditary tendency* is the most important factor in the etiology of gout. It can be traced in about half the cases. If there be an hereditary predisposition, a slight amount of the following exciting causes will induce the disease, while persons without this hereditary tendency are rarely affected by gout, even when they are greatly exposed to the same injurious influences. Gout does not occur during childhood; it is rarer among women than men; in the latter it usually occurs after the thirtieth year of life. Among poor folks it is so rare, that the disease is hardly ever seen in hospital; among the well-to-do classes, it chiefly affects persons given to the pleasures of the table, who drink wine or beer regularly, and take little exercise. All these points render it probable that, next to hereditary predisposition, the supply of more nourishment than is used is to be regarded as the most important etiological factor of gout. Among children, women, mechanics, and poor people, this disproportion is rare; but even during manhood, where it is common among the better classes, those persons escape the gout who do not eat and drink more than is necessary to replace what has been used up in the body. The assertion that in the above mode of life a larger amount of uric acid is formed, because so much of the nitrogenous constituents of the body are broken up, that there is not sufficient oxygen introduced by respiration to transform them into urea by further oxidation, and that this increased formation of uric acid and its collection in the blood appears under the form of gout, is a pure hypothesis which is not supported by facts, although it seems very plausible. If the case were so simple, the world would swarm with gouty patients, while, as it is, only a small proportion of topers and gourmands are affected by gout.

The experiments of *Hoppe-Seyler* and *Zalesky*, who, in order to find out something about the origin of uræmia, tied the ureters in geese and chickens, and, a few days after the operation, found almost all the viscera covered with crystals of the urates and the joints incrusted with them in a manner that very strongly reminded of gout, render the following hypothesis of the relation of the gouty diathesis to the attack of gout the most probable. The gouty diathesis depends on an anomaly of nutrition, in which far more uric acid is produced than in healthy persons, although not to the same extent as in birds and serpents. As long as the excess of uric acid is regularly excreted by the

kidneys, the affected persons get along tolerably well, or have no trouble at all. But the case is different when the uriniferous tubules are plugged up by deposits of the urates, and the excretion of urine is impeded; for then, just as in the animals above mentioned, there is a collection of urates in the blood, and they are deposited in the joints and other organs, and we have an attack of gout. If the deposit of urates in the uriniferous tubules be soon washed away, and the red sandy sediment form in the urine, which we sometimes find even on the diapers of young children, the attack of gout passes over; if this do not take place, and the kidneys atrophy, the acute attack becomes chronic gout. This hypothesis is strongly supported by the fact that *Garrod* and other trustworthy observers have always found the kidneys diseased, they being irregularly atrophied and the seat of uric-acid infarctions. I am far from considering this theory, to which *Traube* also is inclined, as proved; but I think that it agrees with the physiological and pathological facts better than any other theory. If it should finally be proved that the blood of gouty patients always contains more uric acid than normal, while the urine constantly contains less than usual during the attack, our theory would receive a still further support.

Anatomical Appearances.—On autopsy of gouty patients, which is of rare occurrence in hospital, in the affected joints we find the remains of a more or less intense inflammation, and genuine gout is characterized by incrustation of the surfaces of the joint by chalk-like masses, consisting of urates. In mild grades of the disease, only a few joints are changed in this way. Frequently only the first joint of the great toe is in this state. In severe and old cases, we usually find numerous joints diseased; their articular surfaces are covered with thick layers of urates, the cartilages are rough, the ligaments, periosteum, and the mucous bursæ near the diseased joints, are covered with thick deposits of these salts. In such cases the joints are often much deformed, and through the dark, bluish-red skin we may often see white chalky masses. We may also find these deposits at other points, especially in the knuckles, eyelids, and cartilages of the ears. In the auricle, where they are only covered by thin skin, they form white pearls surrounded by varicose vessels (a certain sign of arthritis). I know nothing from personal observation of the above-mentioned changes in the kidney. According to *Garrod*, who has very carefully described and pictured them, gouty kidneys very much resemble those in the third stage of Bright's disease, except that, in the pyramids, there are always white striæ, consisting of urates running in the direction of the tubuli recti, and on the apex of each papilla a white point of the same substance.

Symptoms and Course.—Most gouty patients have shown, by their appearance or sensations, some other results of high living before the first attack of gout. They have usually become fat; the face, particularly the nose, is reddened, from the development of varicose vessels; they have hæmorrhoids; we cannot, however, say that these, and other signs which occur in various modifications, are premonitory tokens of this disease.

But, besides the above symptoms, the first attack, as well as the subsequent ones, is usually preceded by true premonitory constitutional symptoms, arthritis imperfecta, or status arthriticus. The patients feel languid, their sleep is restless; the appetite is impaired, and they have indigestion; they complain of palpitation of the heart and constriction of the chest; they sweat profusely, and frequently pass small amounts of concentrated urine. Careless patients do not generally pay much attention to these premonitions, say nothing about them to their physician, and continue their previous mode of life.

Hence the attack of gout almost always comes unexpectedly, in spite of the premonitions, and surprises the patient like a thief in the night. After he has gone to bed, without dreaming of the coming evil, and has gone quietly to sleep, he is awakened, generally soon after midnight, by a severe burning piercing pain in the metatarsophalangeal articulation of the great toe. The pain rapidly becomes unbearable. The patient feels as if the affected joint were in a vice; he sighs, moans, throws himself around in bed; the leg or even the entire body trembles with pain. Soon after the commencement of the attack, the skin covering the affected joint begins to swell and redden, and there is fever, with a full, bounding pulse, dry skin, intense thirst, concentrated urine, and great mental excitement. Toward morning there is a remission, and in the course of the following day the state of the patient generally becomes endurable, although the pains have not entirely disappeared, and the ball of the affected toe is more swollen, shining, and very red, and the whole leg is slightly swollen by œdema. The next night the scene of the past one is repeated with equal or somewhat less severity; the following day brings another remission, and so passable days alternate with bad nights for about a week, rarely onger, at least in the first attack; then the patient is temporarily free from his trouble. After the redness and swelling have gradually subsided from the ball of the great toe, there is a desquamation of the cuticle, and the parts return to their normal state. The first attack of gout hardly ever leaves any deformity. It does not often affect any joint but the first one of the great toe; in other words, it is rare for the first attack, instead of being a *podagra*, to be a *chiragra*, *gonagra*, or *omagra*. After the patient has recovered from his pain and sleep

less nights, he usually feels better than before the attack; hence the gouty attack has often been considered as critical, and it has been asserted that during or through it a materies peccans has been removed from the body. There is no reason for assuming this hypothesis in explanation of the improved condition. The attack of gout places the patient under circumstances directly opposite to those which have induced his illness. The transmutation of the constituents of the body is greatly increased by the fever, while there is a very insufficient supply of replacing material; the sleeplessness and pain also increase the consumption, or hinder the change of tissue; the disproportion between supply and demand, which we have indicated as the most important etiological factor in gout, and which also lies at the root of its complications, such as corpulence, piles, etc., is thus more than equalized by the gouty attack and the accompanying fever; and this is the simple explanation of the improved condition of the patient after the fit. If pain and suffering that have left no mark were not so soon forgotten, the first attack of gout would often be the last. But, after following their good resolutions and the advice of the physician for a few months, gouty patients generally return to their previous habits; the first attack is followed by a second, this by a third, and so on by a series of paroxysms whose course corresponds with that of the first. At first, the interval between the attacks lasts a year or more; later, several attacks occur in one year. As the intervals become shorter, the attacks usually deviate more and more from the first type; the free intervals are then generally less perfect; the gout, instead of being regular, becomes irregular, chronic instead of acute, atonic instead of tonic.

We give the name of chronic gout to those irregular forms where the attack is preceded for some time by premonitory symptoms, especially dyspepsia, where the seizures are accompanied by less pain and fever, but last for weeks or months, and several joints are affected at the same time, or in succession; and the previously-described chalky deposits of the urates in and about the joints occur chiefly in chronic gout. The swelling and redness, which in acute attacks attain their height the second day, develop more slowly in chronic gout; the redness is generally less intense, the swelling more diffuse and œdematous. After the termination of the paroxysm, the swelling does not disappear with desquamation of the cuticle, as it does in acute gout, but it continues, feels soft and doughy at first, and later it contains firm bodies of variable size, and at last a hard tophus is left. This is small in proportion to the tumor whose remains it represents, but after repeated attacks it grows by new deposits, and may attain a considerable size. The deposits within the joints, the calcareous masses as well as the inflammatory changes in the capsule and ligaments of the joint, induced

by the continued irritation of the deposits, after a time cause pain, difficulty of motion, and deformity of the affected joints during the interval between the attacks. The patients often have difficulty in getting about with the aid of a cane, and the usefulness of the hands and arms is impaired if these be affected. In some cases, irritation of the surrounding parts, by the concrements, induces phlegmonous inflammation. Abscesses form about the joint, and mortar-like masses, or firm concrements, are not unfrequently mingled with the pus evacuated from them. In proportion as the function of the limbs is gradually impaired in chronic gout, the patient becomes infirm. The fulness of body and strength are lost, and the digestion is impaired; the patient suffers from acid stomach, flatulence and irregular bowels; often, also, there is disturbance of the circulation, and generally great irritability and uneasiness. It is customary to refer the numerous disturbances of function and nutrition that occur in the course of chronic gout to the anomalous condition of the blood, or the gouty dyscrasia; still it is possible that an insidious fever, only to be recognized by the thermometer, accompanies chronic gout, and induces the general cachexia. As a rule, chronic gout results from the acute form, as we said above, after the patient's constitution has suffered from repeated attacks of the latter, or from debilitating treatment; but cases also occur where the disease is primary.

Atonic or *anomalous* gout is the designation generally applied to those forms where there is no actual attack of gout; or, as the name "atonic" indicates, where the debilitated organism is not in condition to develop a normal attack of gout. Formerly the diagnosis of atonic or anomalous gout was carried to excess; but, on the other hand, it is certainly going too far to class many of the symptoms, some of them very peculiar, as accidental complications of the disease, and not directly dependent on it. In persons who have previously suffered from attacks of regular acute gout, we not unfrequently meet a condition which we are perfectly justified in calling atonic gout. In such patients there is a permanent constitutional affection, which shows itself chiefly by excessive general hyperæsthesia, muscular weakness, and dyspepsia, and usually also by an increased amount of perspiration, and thick, cloudy urine. The most insignificant causes, such as slight errors of diet, excitement, exposure to cold, changes of weather, and often without perceptible cause, these constitutional symptoms are accompanied by pains in one or more joints, which are very severe, and resemble commencing attacks of gout. But there is only a moderate redness and slight swelling, which disappear after a few hours; in other cases there is no redness or swelling, and the severe pain is the only indication of the gouty attack. If there have already been

ordinary attacks of the disease, this not unfrequent condition is readily recognized, and intercurrent acute attacks place the diagnosis of atonic gout beyond doubt.

The correct appreciation of the symptoms, and the diagnosis of the disease from chronic wandering rheumatism, is far more difficult, if there have been no precedent or intercurrent attacks of gout. It seems to me there is no doubt that this atonic form of the disease is also occasionally primary, and even when there have been no attacks of gout I think it must be diagnosed, where there is an hereditary predisposition, and where the disease has been preceded by luxurious living, and where it is chiefly the smaller joints of the foot and hand that become painful, red, and swollen. I have seen such cases where the diagnosis was strongly supported by the experience of the patient. The patients themselves have not only remarked that they could expose themselves to catching cold with impunity, while the return to careless diet, and the use of beer and wine, always made them worse; but they have also observed that antirheumatic treatment did not improve their case, while there was actual and permanent improvement both of the local symptoms and general cachexia, by a course of treatment hastening the transformation of tissue.

I have no doubt that in gouty patients there is sometimes a deposit of urates, accompanied by hyperæmia and inflammation, in other organs instead of in the joints; or, in other words, that an anomalous internal gout, the *arthritis metastatica retrograda* of the old authors, actually occurs. For the support of this view, I lay less weight on the observations of *Zalesky*, who, after ligating the ureters of chickens and geese, found deposits of urates not only in the joints, but in almost all the organs, among others in the stomach, heart, and lungs, than on a series of clinical observations, which admit of no other interpretation. For instance, some time ago, in an old gentleman, who had suffered for years from gout, I saw an angina begin simultaneously with a typical attack of gouty inflammation of the joints; it was characterized by a peculiar blue color of the fauces, by a very different course from ordinary forms of angina, and lasted as long as the inflammation of the joints did. I have not the slightest doubt that this was a case of gouty angina. In two other patients, who also suffered from gout, I have seen cerebral troubles, which I must regard as circumscribed gouty inflammation of the meninges. All other diseases of the brain or membranes could be excluded with certainty, especially when the symptoms, which appeared very dangerous, and had excited great anxiety, disappeared in the one case with a copious excretion of urates in the urine; in the other, with an attack of gouty inflammation of the joints. It is true, autopsy has not proved the occurrence of in-

ternal gout. But we must remember how rarely we have the opportunity of making autopsies on gouty patients, and how carefully an examination would have to be made to discover deposits of urates in the gastric mucous membrane, lungs, or heart; also, that it is not at all improbable that these deposits disappear after the attack is over. The organs most frequently attacked by gout appear to be the stomach, brain, and heart. 1. *Gout in the stomach* appears with the symptoms of severe cardialgia, which is occasionally accompanied by violent vomiting, and sometimes with hæmatemesis. 2. *Gout in the brain* may sometimes present the appearance of apoplexie foudroyante, while in other cases it manifests itself by severe circumscribed headache, dizziness, and vomiting. 3. *Gout in the heart* induces irregular and enfeebled action of that organ, and, as a consequence of the disturbed circulation, dyspnœa, or fainting. Lastly, cases are reported where there was metastasis to the spinal marrow, inducing sudden paraplegia, and to the lungs, causing asthmatic attacks. We must take care not to refer every intercurrent disease, affecting a patient of gouty diathesis, to an attack of anomalous gout. We are only justified in so doing when the disease runs its course with unusual symptoms; also when the patient is affected with gouty inflammation of the joints at the same time; when disease, running a peculiar course, occurs suddenly in an internal organ while a gouty affection of the joints is subsiding or developing; and lastly, when the termination of the disease is accompanied by a copious excretion of urates through the kidneys.

The course of the disease is tedious and insidious. The termination in permanent cure is rare, but would be more common if the patient would entirely change his mode of life before the disease becomes firmly rooted. Death, also, is a rare termination, and results almost exclusively from severe attacks of anomalous internal gout. Most patients die of complications, or of intercurrent diseases.

Treatment.—Since, next to hereditary predisposition, the disproportion between supply and demand is the most important cause of gout, the causal indications require the removal of this disproportion by limiting the supply and increasing the consumption. During the course of gout, particularly when the disease has become irregular instead of regular, there is a time when the state of the patient does not permit a restriction of the supply, and where the increase of consumption must be made very carefully; but in all recent cases of regular gout, and in all patients whose general health and nutritive condition indicate a continuance of this disproportion, the fulfilment of this indication is the only means from which we can expect a cure. Hence we see that in treating gout we should write few prescriptions, but

should regulate the habits of the patient. First, it is necessary to prescribe precisely the form, quantity, and quality of his food. A gouty patient should be carefully taught what and how much he may eat, because, although he breaks the rules often enough, he gives way to excess less readily than he would if he could excuse his errors by the plea of ignorance. How many patients there are who fear the evil results of breaking the rules laid down for them far less than they do the reproaches of their physician! Gouty patients must be absolutely forbidden to go to dinners, etc., even if they promise to be very moderate. It is best for them to eat only vegetables, soups, etc., and to have meat only once a day. The use of beer and wine retards the transformation of tissue, and hence is injurious for gouty patients. Any one may observe on himself that he requires less to eat when he drinks wine or beer with his meals, and also that when using these articles moderately he bears fatigue better than he does without. Persons but little inclined to the production of fat, under the regular and free use of wine and beer, become very obese, and from this mode of life most persons have a red face and distended veins, until their digestion is impaired or there is some other injurious consequence. This consideration, as well as the fact that in persons who drink neither wine nor beer the occurrence of gout is very exceptional, should induce us to forbid these drinks altogether, or else to give directions to have them stopped off gradually. The same is true of the use of tea and coffee. Although there is but little nutritive material in these drinks, and consequently the actual supply of nutriment to the body is but little increased by their use, still there is no doubt that tea and coffee have the same influence on the transformation of tissue that wine and beer have; they preserve the strength, diminish the need of nutriment, limit the consumption, and hence are injurious to gouty patients. Drinking large quantities of water has just the opposite effect on the transformation of tissue from what is induced by the use of tea and coffee, or of beer and wine. After drinking freely of water, no one feels less need of food, fatigue is not better borne, it does not induce corpulence and a red face; on the other hand, it has been shown that, when plenty of water is taken, the amount of water passed is greater than it would be under like circumstances, without this increased supply; and as the amount of urea excreted after drinking plenty of water is increased permanently, not temporarily, we are led to the conclusion that much water hastens the transformation of tissue, and hence is as beneficial for the gouty patient as drinking coffee and tea or wine and beer is injurious. Lastly, since muscular action also hastens the transformation of tissue and the consumption of the constituents of the body, it may be readily seen that a lazy, easy life is

bad for gouty patients, and that active exercise is important in the treatment. We should not have entered into this discussion of theories, if theory and practice did not fully agree in the treatment. The rules that we might lay down for the treatment of gout, from the known action of certain substances on the transformation of tissue, have long since been proved correct at the bedside of the patient. The treatment by mineral waters forms a kind of connecting link between dietetic and medical treatment; it is perhaps more highly esteemed in gout than in any other disease. The springs celebrated as being antiarthritic are those of Vichy, Karlsbad, Marienbad, Kissingen, Homburg, etc. The favorable effect of these mineral waters appears to depend on their reducing the plethora, due to a misproportion between supply and demand in the body, whether the plethora depend solely on hypertrophy of the blood, i. e., an increase of its cellular elements and a certain density of the intercellular substance (the serum of the blood), or on an accompanying absolute increase of the amount of blood contained in the body. It is very interesting to note that the beneficial influence of these natural mineral waters on plethora, which has been long known, and which far exceeds that of ordinary water, agrees with the observations of *C. Schmidt* and *Vogel*, according to which the amount of albumen in the serum of the blood is inversely proportional to the amount of salt. I am undecided as to which of the above springs deserves the preference in the treatment of gout, whether the solution of salt, of which the Kissingen and Homburg waters consist, removes the plethora more rapidly and completely than Karlsbad and Marienbad water, or the reverse. Nor shall I attempt to say whether the supply of those solutions of salt act beneficially not only on the plethora but also on that anomaly of the change of tissue which shows itself as the gouty (uric acid) diathesis in some plethoric persons. Nor can we, with our present knowledge, say whether in any particular case the preference should be given to Kissingen, Karlsbad, Wiesbaden, Homburg, or Vichy, and what would constitute the peculiarity of the case which indicates one rather than the others. It cannot be denied that in recent times the regular therapeutic employment of the so-called *Bullrich's* salt, a mixture of bicarbonate and sulphuret of soda, rivals the world-renowned success of these springs—a fact which is at least opposed to the asserted latent peculiarities and advantages of the natural solutions of salt. Advantageous as the above treatment proves in recent cases of regular gout, if carefully and judiciously instituted, there is often great harm done by excessive limitation of the supply of nourishment, by the sudden complete abstraction of spirituous liquors that had been used for years, as well as by all other debilitating courses of

treatment which are employed carelessly or hastily. The patient is often freed from attacks of acute gout by this too zealous or too hasty treatment, but becomes affected instead with irregular, chronic, or atonic gout, an exchange by which he certainly gains nothing. As soon as gouty patients begin to show the signs of general cachexia, their disease is rendered worse by a continuation of this debilitating treatment; we should then prescribe nutritive diet for them, and even allow them to drink wine. But we must warn against excess even in the strengthening treatment then indicated. Although in this stage of the disease it is not admissible to limit the supply of nutriment, we should nevertheless carefully try to increase the change of tissue. Under no circumstances should we allow the patient to give himself up to sluggish ease, but should see that he exercises as much as his strength permits; we should not allow more wine than is sufficient for a slightly tonic and refreshing effect, and it is always better to let the patient use the ferruginous, alkaline-saline and alkaline-muriatic mineral waters, such as Eger, Kissingen, or Homburg, than to prescribe simple chalybeate waters or preparations of iron. If there be at the time no indication for prescribing solutions of salts, we should at least see that the patient drinks plenty of water. In order that this prescription may be regularly obeyed, we should direct how much water is to be drunk daily. The probability that a retention of urates from obstruction of the uriniferous tubules induces the attack of gout, makes it appear practical to maintain the secretory pressure in the kidneys at a certain height, and to dilute the urine, as the urates require a great deal of water for their dilution. In the later stages of the disease the akratothermal springs, Wildbad, Gastein, Pfäffers, etc., are very serviceable. We may have the patient drink of these waters and bathe in them; perhaps the infarctions obstructing the tubules may be carried away by the former, and the kidneys washed out, as it were, while the latter has the most beneficial effect on the inflammation of the joints.

We cannot give any rules for fulfilling the *indicatio morbi*, as the gout is a peculiar and obscure disease, which we can neither cure by so-called rational treatment, nor have we any specific for it. It is true some physicians consider colchicum as a specific in gout; but its action appears to be only palliative. The practice of giving colchicum for a long time is being gradually discontinued, and it is administered only during the acute attacks. Most physicians of the last century, who generally had a high opinion of the efficacy of medicines, considered gout as a *noli me tangere*, and even at present we cannot sufficiently warn against the uncalled-for and injurious administration of medicaments in this disease.

The symptomatic indications require us, first of all, to shorten the attack and to render it as bearable as possible; for the idea that the gouty attack must be guarded and cared for, because it has a beneficial depurative effect on the organism, has been very justly abandoned. Experience has shown that, in gouty inflammation, antiphlogistics neither alleviate the pain nor shorten the attack; and it has also shown that the untimely use of antiphlogistics, especially general and local bleeding and active saline purges, favors the passage of regular acute gout into irregular chronic and atonic gout. We should also advise against the application of cold or of warm and narcotic compresses over the painful joints; on the other hand, in attacks of acute or chronic gout, the narcotics, and, above all, colchicum, should be freely given. It has not yet been decided to what peculiarity colchicum owes its efficacy. *Garrod* has shown that it is not chiefly due to its diuretic power and the removal of the collected urates from the body. It is customary to give twenty to thirty drops of the tincture, or vinum seminum colchici, four times daily. These doses do as much good as larger ones, which cause pain in the abdomen, and diarrhœa. Besides the colchicum, it is well to give the patient plenty of acid drink during the attack; perhaps the favorable effect of this treatment depends, as I above indicated, on the dilution of the urine and the increased lateral pressure in the kidneys. Our theory would also agree with the treatment of the attack after the method of *Cadet de Vaux*, according to which the patient is to drink six ounces of simple water, as hot as possible, every fifteen minutes. It really seems as if this were of some benefit, although it is not altogether free from danger. During the attack the affected limb should be elevated and covered with cotton or wool, and the patient placed on low diet. If the motions be impaired after the attack, treatment by baths in Wildbad, Teplitz, or Wiesbaden, is indicated. If abscesses form near the joints attacked by gout, we should use cataplasms; if the abscesses lead to ulcers, the applications should if possible be continued till the ulcers have healed. No general rules can be given for the treatment of attacks of anomalous internal gout. Abstraction of blood readily induces dangerous collapse, and, on account of the threatening paralysis, stimulant treatment is usually more appropriate. If the disappearance of a peripheral affection be followed by an attack upon the brain, stomach, or heart, we may cover the part previously affected with irritating and vesicating plasters, although this rarely does much good.

CHAPTER VI.

RACHITIS—RICKETS.

Etiology.—The actual changes in the bones in rachitis consist, 1. In proliferation of the cartilage of the epiphysis and of the periosteum, which are the sources of the normal growth in length and thickness of the bones. 2. The cartilaginous and fibrous tissues, resulting from this proliferation, ossify more incompletely and later than in the normal growth of bone. Hence, in rachitis, there is not, as was formerly supposed, a morbid softening of tissues previously hard; but tissues which normally become hard, from deposits of chalky salts, remain abnormally soft. This view is not in opposition to the fact that rachitic bones bend more readily than they did before the disease. The medullary cavity increases in rachitic just as it does in healthy bones; but, while in the latter the new formation of firm, bony substance at the periphery preponderates over its loss from within, so that, in spite of the latter, the strength of the bone increases; in rachitic bones, the loss of firm, bony substance from within is not replaced by a corresponding new formation at the periphery, and consequently their resisting power is decreased. The excessive proliferation of the cartilages of the epiphysis and of the periosteum, with which rachitis begins, is by some regarded as inflammatory. The great vascularity and infiltration of the affected parts, the pains which accompany the disease from the first, as well as the many analogies of the proliferation, with other undoubtedly inflammatory affections, support this view; but, on the other hand, it is opposed by the etiology, course, and constant results of the disease. Many attempt to explain the retarded ossification of the newly-formed cartilage elements, and the fibrous proliferation of the periosteum, by saying that, in rachitic children, the calcareous salts taken up with the food cannot be deposited in the terminal and peripheral layers of bone, because they are held in solution by the lactic acid in the blood of the patient, and are excreted through the kidneys. Part of the analyses of urine made favor this explanation, as they showed that the urine of rachitic children was not unfrequently very rich in lactic acid, and that it contained four or six times as much phosphate of lime as is contained in normal urine. Another point in favor of this view is, that children who suffer from dyspepsia are especially apt to be attacked with rachitis. The active decomposition going on in the stomachs of these children forms quantities of acid, particularly lactic acid, and it is possible that their reabsorption and their presence in the blood hold the phosphate of lime in solution, and that by their excretion with the

urine, the material for the formation of bone-substance is withdrawn from the blood. But this hypothesis is not tenable; the increase of lactic acid and phosphate of lime in the urine of rachitic children is not constant. Rachitis not unfrequently develops without precedent disturbance of the digestion or formation of acid, and the proliferation of the epiphyseal cartilages and of the periosteum, which are just as important factors of rachitis as the retarded deposit of calcareous salts is, cannot be explained by the excretion of phosphate of lime through the urine. *Virchow*, to whom we owe the most important explanations of the histology of rachitic bones, and the pathogeny of rachitis, says that the cause of the retarded ossification is more probably a diminished supply of chalky salts than their increased excretion through the urine. He reminds us of the recommendations of the carbonates and phosphates of lime as remedies in rachitis, and calls particular attention to the fact that, in the dyspepsia of children, which usually precedes rachitis, as there is less albuminate taken into the system, the amount of earthy salts required for the normal growth of the bone absorbed is also diminished, as they are, for the most part, introduced into the body as albuminate. *Virchow* does not pretend that this view also is not problematical. It does not explain why the disturbance of the supply of nutritive material should affect the bones more than the other parts; it is also opposed by the fact that rachitis occurs not only in dyspeptic children, but also in those who are otherwise well nourished; lastly, it is evident that the diminished supply of nutritive material does not suffice to explain also the proliferation of the epiphyseal cartilages and of the periosteum. From these considerations, it seems to me that the most probable hypothesis regarding the cause of rachitis is that which refers it to inflammation of the epiphyseal cartilages and periosteum. In other tissues, also, as in the skin, mucous membrane, etc., we often see diffuse inflammations (exanthemata, catarrhs, etc.), whose immediate cause we cannot discover. Like rachitis, these occur chiefly, but not exclusively, in badly-nourished, cachectic persons, and are also often accompanied by rachitis. From what we know of periostitis, it is not surprising, as *Virchow* aptly says, that the disturbance of circulation at the height of the disease should hinder the deposit of calcareous salts in the morbidly-proliferating tissue. Lastly, the excessive excretion of phosphate of lime through the urine may just as well be the result as the cause of the lime-salts not being deposited in the bones. Rachitis is a disease of childhood, and it is doubtful whether the rare cases, where the disease is said to have occurred in adults, or during fœtal life, were actually rachitis. The disease is most frequently seen from the second half of the first year till the time of the second dentition; before the seventh month,

and after the seventh year, it is far more rare. In some families, the tendency to it appears to be hereditary. Improper nutrition of children is undoubtedly the most frequent cause of the disease. I also believe that the gastric and intestinal catarrh, due to the improper nutrition, greatly favor the occurrence of the disease, although I do not consider it as proved that this results from reabsorption of the lactic acid. The occurrence of rachitis in well-nourished children, with unimpaired digestion, shows that, besides the above, there are some other unknown causes of the disease.

Anatomical Appearances.—For the histological details of the examination of rachitic bones, I refer to the works of *Virchow*, *Kölliker*, and *H. Mayer*. *Virchow* enumerates the changes observed in the epiphyseal cartilages during rachitis as follows: 1. Arrest of the line of ossification while the preparatory line of proliferation of the cartilage relatively increases. 2. Encroachment of the medullary space into, or even beyond, the line of ossification, while the proliferation of cartilage continues. 3. Formation of fibrous medullary spaces, osteoid transformation of the parts around them, as well as of distant parts, with calcareous deposits. The processes observed in the diaphyses are grouped as follows, by *Virchow:* 1. Greater density of the periosteal proliferation, with progressive rarefaction of the substance in the areolæ and cancellated tissue. 2. Deficient ossification of the cancellated tissue, and continuance of the deep layers of compact exterior substance. 3. Partial formation of cartilage in the areolæ.

The clumsy appearance of rachitic bones and the swelling of the epiphyses are sufficiently explained by the proliferation of the periosteum and epiphyseal cartilages. The epiphyses are thickened and not elongated, according to *Virchow;* not because the proliferation is chiefly lateral, but because the soft proliferating layers are compressed, and deflected laterally by the weight of the parts pressing on them, and by muscular action. The distortions of rachitic bones depend partly on curvatures, partly on angular deformity. The curvatures occur chiefly at the epiphyses and at the points of cartilaginous union of bones which have no epiphyses, while the angular deformities occur chiefly at the diaphyses. In the long bones of the extremities it often looks as if the epiphysis had slipped past the diaphysis. Curvature of the posterior ends of the ribs of one side of the thorax not unfrequently induces asymmetry or obliquity of the thorax. In many cases the points of union between the anterior ends of the ribs and the costal cartilages are bent inward, while the sternum, with the sternal end of cartilages, is pressed forward. This deformity, *pectus carinatum seu gallinaceum*, is due to the softness of the parts mentioned, they having lost the power of resisting the pressure of the atmosphere

by inspiratory dilatation of the thorax. Any point of the ribs where an elastic ligament replaces the bone would become depressed on inspiration. Curvatures of the upper and lower ends of the individual vertebræ cause distortion of the spinal column; curvatures of the pelvic bones at their points of union cause pelvic deformities; the most frequent form is the so-called rachitic pelvis, where the antero-posterior diameter is shortened, but occasionally, also, when the curvature does not occur at the point of union of the sacrum with the ilion, but at that of the pubis with the ischium, we have a heart-shaped deformity. In the angular deformities the bone is only curved on the convex side, but on the concave side it is actually broken. The medullary cavity of a sharply-bent bone is, like the cavity of a sharply-bent quill, greatly contracted at the bent portion, and is subsequently entirely closed by callus. Total fractures, also, are not rare in rachitic bones, but they differ from fractures of healthy bones, in never being accompanied by injury of the periosteum, which is separated from the compact bone-substance by a soft, incompletely-ossified layer. The sutures of the skull, which correspond to the epiphyses of the long bones, ossify very late; hence, in rachitic children even two or three years old, we not unfrequently find the fontanelles still open, and so large as to induce the suspicion of chronic hydrocephalus. The bones of the head and face, which correspond to the diaphyses of the long bones, undergo the same changes as the latter do; imperfect ossific proliferations of the periosteum occur on the bones, especially near their edges, and deform the head and face. Besides this thickening, a partial thinning of the cranial bones is also observed in rachitis (*Elsässer's* soft occiput, craniotabes). This thinning, which may finally let the dura mater and pericranium come in contact, results from gradual disappearance of the cranium under the pressure of the gradually-increasing brain, which is not accompanied by coincident formation of new bone-substance on the surface of the skull. These membranous spaces are most frequently observed in the occiput, more rarely in the parietal or frontal bones; they occur chiefly at places where the impressiones digitatæ, corresponding to the convolutions of the brain, are normally found. A symptom analogous to craniotabes is also seen in the lower jaw, the anterior wall of the alveolus being often perforated by the milk-teeth. If the rachitis goes on to recovery, the swelling of the articular ends of the bones subsides; the bones become firm, while only a small amount of the curvature of the limbs is corrected; after the disease has run its course, the cartilaginous proliferations of the epiphyses often ossify sooner and more completely than is desirable for the longitudinal growth of the bones, for which it is necessary that the epiphyses should remain cartilaginous. It is, probably, on this ac-

count that persons who have suffered from extensive rachitis usually remain very small, or even dwarf-like. If certain bones only have been rachitic, while others have escaped, a marked disproportion remains after the disease has subsided, from the irregular growth of the bones. The shortness of the extremities has much less effect on the general health than the distortion of the thorax or the rachitic pelvis, which are also due to the slow growth of the thoracic and pelvic bones after rachitis. Since the sutures and fontanelles of the skull remain open for a long time, and since, until the sutures are closed, the growing brain does not permit any distortion of the skull, the growth of the skull is not arrested, and we often find persons, formerly rachitic, very much deformed, having a small face surmounted by a large head, out of all proportion to the misshapen body.

Symptoms and Course.—Rachitis occurring during the first months of life is so often preceded by chronic intestinal catarrh, with stools at first green and mucous, subsequently copious and watery, that it appears almost justifiable to regard this form of chronic intestinal catarrh, the "diarrhœa ablactatorum," "yeasty diarrhœa of children," among the prodromata of rachitis. But the fact, that numerous cases of this chronic intestinal catarrh do not pass on to rachitis, but sometimes remain uncomplicated, sometimes induce other disturbances of nutrition, renders this view doubtful, as well as that of *Stiebel*, according to which, "cacotrophia" is the first, muscular atrophy the second, and disease of the bones the third stage of rachitis. If, after the above state has lasted for a time, we cannot find any changes in the bones during life or on autopsy, there is hardly any proof that the disease is rachitis. The first symptom, showing that rachitis has succeeded the diarrhœa and consequent marasmus, is the pain that the children unmistakably suffer when they attempt to move their limbs, or when they are moved by any one. *Stiebel* very truthfully describes how children, whose greatest pleasure had been to kick out their legs and put their toes in the mouth, then lie quiet, with their thin legs held straight out, as if afraid to move; how they cry at every motion, and even begin to whimper for fear of being taken out of the bed when persons, that they had formerly loved, approach them. These symptoms are succeeded by enlargement of the epiphyses—which is most noticeable at joints not thickly covered by soft parts, or normally very prominent—as the knee and elbow are, and in the lower epiphyses of the radius and ulna, as well as at the point of union of the ribs with their cartilages.

If rachitis begin in the manner above described, from diarrhœa ablactatorum, that is, if it begin when the child has not yet attempted to walk, it often escapes any distortion of the extremities, even if the disease last for years. This shows clearly that the curvatures and partial

fractures of rachitic bones are chiefly due to the weight of the body resting on them, and to the action of the muscles. We have given a different explanation for the occurrence of pectus carinatum; and the fact, that children who have become rachitic during their first year, and still have straight legs, usually have chicken-breast, perfectly agrees with this explanation. Children attacked by rachitis during the first year are the ones who are affected with craniotabes, from lying so long on their backs, probably from the pressure of the brain on the inner surface of the skull, while the bed presses on the outer surface. It is of little importance whether the rolling the head, thinness of the hair over the occiput, disturbed sleep, attacks of spasmus glottidis, eclampsia, and other symptoms of anomalous cerebral functions, which are often seen in such children, are to be regarded as the results of craniotabes, or as concomitant symptoms. Careful pressure on the soft parts of the head is well borne by many children, while in others it induces convulsions. We must further mention that chronic bronchial catarrh is the most frequent complication of rachitis early in life; its absence is exceptional. In this disease, the teeth are generally cut very late, and often irregularly; it frequently happens that the child is a year old before he has a tooth in his mouth. Finally, there is no question that rachitic children, who have acquired their disease during the first months of life, usually differ from other children of equal age, by their bright answers, and their attentive, sensible manners; but, in spite of the large head, we cannot agree to refer this precocious mental development of rachitic children to hypertrophy of the brain, because we also see it in other children who are confined to the bed by any disease, and so kept away from other children, and constantly surrounded by sedate, grown persons; and because in so-called hypertrophy of the brain there is not an increase of the nerve-elements, but of the neuroglia. Moreover, the contrast between the physical helplessness and the mental development causes the latter to appear greater than it really is; and children three years of age, who cannot yet walk, are usually considered younger than they really are. If the disease end in recovery, the first sign of improvement is generally a decrease of the emaciation. The loose skin is again filled by the limbs, the wrinkled and old face grows smooth, while the protuberant belly becomes smaller. After a while the children begin to sit up in bed, and play. At this time there is great danger of curvature of the upper and lower ends of the vertebræ, and permanent distortions of the spinal column. At the commencement of convalescence, curvatures and partial fractures of the extremities also are most likely to occur when the children attempt to get out of bed, and walk around, holding on to the chairs. The symptoms of rachitis differ in some

points from the above description, when the disease occurs in older children. In them the symptoms of chronic intestinal catarrh, and general emaciation, do not usually precede the symptoms of rachitis; the children often have, apparently, normal digestion, and are well nourished when the disease develops. Nor do they usually suffer the pain that is induced in smaller children by all active and passive movements. They only complain of pain when they make a misstep, and easily become fatigued. Finally, when the children attacked by rachitis are two or three years old, or older, the ribs and vertebræ escape in the commencement, and the extremities first become deformed. They are affected with curvatures, and partial fractures, whose direction is not always the same. Sometimes they form an increase of the normal curvature of the bones; sometimes they are in the opposite direction, variations whose cause is unknown. Not unfrequently the thighs are bent outward, the legs inward; the children acquire a clumsy, waddling gait. It is generally a long time, often many years, before the disease extends to the whole skeleton. Then we notice frequently, but not always, as *Guérin* asserts, that the disease extends with a certain regularity; first, the bones of the legs, then of the thighs, later those of the forearms, of the arm, and lastly those of the trunk, are attacked. Even when we succeed in arresting the disease at an early stage, the small size, the misshapen limbs, as well as slight curvatures remaining in the lower extremities, remind us through life of the rachitis that occurred during childhood; in severe cases very unsightly curvatures and shortenings of the bones remain, which not unfrequently impair the functions of the body.

Treatment.—In cases where the rachitis develops from chronic intestinal catarrh and marasmus, the causal indications require the treatment described in Vol. I. We there remarked that it was difficult to arrest the active process of decomposition going on in the intestinal canal of children, which induces the diarrhœa, and we must consider the want of success in the treatment of this diarrhœa as the chief cause of the tardy and imperfect results of treatment in rachitis. If we succeed in curing the intestinal catarrh speedily and completely, and in improving the nutrition of the child, the rachitic symptoms, induced by these disturbances, also generally disappear in a short time; and, if the disease be recognized early, the children escape any permanent results of the rachitis. In the treatment of irritative diarrhœa, if it be accompanied by rachitis, in view of the diminished deposit of chalky salts in the tissues, it is customary to prefer carbonate of lime, in the form of prepared chalk, to the carbonates of potash and soda, which are generally used. In other cases, where the disease occurs without our being able to discover chronic intestinal catarrh, or other

disturbance of nutrition as its cause, we cannot fulfil the causal indications.

The indications from the disease are not answered by giving carbonate and phosphate of lime, for the absence of the bony earths from the bones is not the chief anomaly in rachitis. As soon as the disturbance of nutrition in the epiphyseal cartilages and in the periosteum has ceased, the ossification of the proliferation begins, and, indeed, often becomes excessive, even if no material for aiding ossification be supplied, except the calcareous salts taken in the ordinary articles of food. Bitter and tonic remedies also, as well as rubia tinctorium, have not fulfilled the hopes entertained of them on theoretical grounds, and have been almost superseded by cod-liver oil, which is very efficacious in most cases where it can be taken, as it generally can be. We do not know to what peculiarity cod-liver oil owes its apparently specific effect in rachitis. Brine baths are also of unmistakable benefit, and should be used, particularly where the children are in a fair condition as regards nutrition, or are even moderately fat. The diet suitable for scrofulous patients, consisting of a small supply of vegetables, with plenty of animal food, is also very important for the successful treatment of rachitis. We would particularly recommend the administration of a moderate amount of finely-shaved raw beef twice a day, followed by a small spoonful of Tokay or Malaga wine. Finally, we should take care that rachitic children be not kept in damp, dark rooms, but sent out of doors as much as possible.

The symptomatic indications are, to guard against curvatures of the bones, and to rectify any existing curvatures. The former indication is more readily fulfilled than the latter; the means for fulfilling it consist, as may be inferred from what we have already said, of the mode of origin of the curvatures and partial fractures, in carefully protecting the soft, pliable bones from the weight of the body, the action of the muscles, and external force. Rachitic children should sleep on mattresses, not on feather beds. It does not make so much difference about the material in the mattress as about the care with which it is made, for on this depend its regular fulness and the evenness of its surface. High pillows should be forbidden. The nurse should carry small children out in a basket; larger ones should be drawn about in the open air, in a carriage provided with a mattress. Until the bones are consolidated, sitting up in bed for a length of time should be prohibited, and still more should we forbid the children running about on their flexible, fragile legs. But if the curvature has already occurred, we should send the child to a reliable orthopædic institute, that is, to one where the actual results after the treatment correspond to some extent with the promises made before its commencement, and the accounts

given while it is being carried out. Of course, institutions where some particular plan of treatment is not followed exclusively, or with partiality, but where all the aids of orthopædia are employed, and especially where the general health of the patient is attended to, should be preferred to others.[3]

CHAPTER VII.

OSTEOMALACIA.

Etiology.—In osteomalacia, bones which have been hard become soft from reabsorption of the salts of lime, to which they owed their hardness. This explanation of the disease at once shows its difference from rachitis, where the lime-salts do not disappear from the bones, but have never been deposited there. The immediate causes of the solution and reabsorption of the salts of the bones in osteomalacia are obscure. The hypothesis, that the appearance of an acid in the tissue of the bones lies at the root of the processes by which the phosphate of lime is dissolved, is refuted by *Virchow*, who found the gelatin escaping from fresh osteomalacic bones was of alkaline reaction. There is a great deal in support of *Virchow's* theory that osteomalacia is, perhaps, to be classed among the parenchymatous inflammations where no interstitial exudation is deposited, but where the inflammatory disturbances of nutrition affect the tissue-elements of the affected organ. The rarefaction, the porous, spongy, or areolar condition of these bones, which are perfectly analogous to the changes in osteitis proper, the frequent occurrence of the disease in the puerperal state, its customary origin from the pelvis, which has been injured during parturition, and lastly, the severe pain accompanying it, render it very probable that osteomalacia is of an inflammatory nature. This is a rare disease; up to the present time it has been observed almost exclusively in women. The exciting causes are unknown; we only know that the first signs of it, in most cases, appeared some time after confinement, so that pregnancy, parturition, or the puerperal state, undoubtedly have much to do with its etiology.

Anatomical Appearances.—In osteomalacia, the bones become very porous; in the spongy substance, the trabeculæ decrease in number, and disappear; hence the medullary cavities coalesce, and in the long bones induce enlargement of the medullary spaces; in the compact cortical substance, also, the vascular channels increase, and form areolæ, which unite and change the cortical substance also into a coarse, meshed, spongy tissue. Finally, in the highest stage of the disease, the soft, flexible bone, which may be readily cut, consists only of

periosteum and medulla, with a few delicate, bony trabeculæ. In recent cases the medulla is dark, brownish-red; later, it is yellow, and very fatty. There are cases of osteomalacia, where the disease is confined to certain bones, especially the pelvis and spine; in other cases it affects almost the whole skeleton; but, even then, the bones of the skull almost always escape. Distortions of the body and limbs result from the softness and flexibility of the bones; these are often very great, and actually frightful. The weight of the head occasionally induces angular curvature of the cervical portion of the spinal column; the distortion of the dorsal vertebræ, and the ribs, may not only cause deformity of the thorax, but may also greatly encroach on its capacity. The pelvis is almost always compressed laterally by the thigh-bones, often to such a degree that the horizontal branches of the pubis come in contact, and project like a beak. The sacrum is also generally curved into the pelvis. Sometimes the curvatures of the extremities attain such a grade, that the feet are at last directed outwardly. In some cases the shortening of the body is so decided, that women of stately size become quite dwarf-like during the disease.

Symptoms and Course.—The first symptoms of osteomalacia are boring, tearing pain, which the patients often locate in the bones. This pain is usually relieved by perfect quiet, while it is increased by motion. In some, but not in all cases, the pain, which at first is usually considered rheumatic, is accompanied by a remittent or intermittent fever. According to many observers, copious sediments of phosphate of lime form in the urine of the patient; where they do not occur, renal calculi, consisting of phosphate of lime, are found after death. It is also said that lime-salts are excreted through the salivary glands and skin. Gradually, the gait becomes uncertain and tottering, and, after a time, any motion becomes so painful, that most patients fear leaving their bed. The pain is followed by distortion and deformity of the body and limbs, which vary from accidental circumstances. Occasionally, the well-nourished state of the patient contrasts for a long time with the pain and deformity; while in other cases the general health is affected early, and the patients appear miserable, and badly nourished. Osteomalacia has not ended in recovery in any of the cases so far reported; but in most of them it was not till after several years of severe suffering that death occurred, from exhaustion, or from disturbances of the circulation and respiration.

Treatment.—The remedies theoretically recommended for osteomalacia, such as calomel and opium, phosphoric acid, lime-water, cod-liver oil, and iron, have not answered in practice, and we are, unfortunately, obliged to confine ourselves to the prevention of distortions.

CHAPTER VIII.

PROGRESSIVE MUSCULAR ATROPHY—ATROPHIE MUSCULAIRE GRAISSEUSE PROGRESSIVE—PARALYSIE MUSCULAIRE PROGRESSIVE ATROPHIQUE.

[*Friedreich* ascribes progressive muscular atrophy to chronic inflammation of the perimysium, inducing hyperplasia of the interstitial connective tissue and destruction of the muscular filaments. But of late the more perfect means for examining the spinal cord show changes in its gray anterior horns, such as atrophy, increased pigmentation, or even disappearance of its ganglion-cells. Apparently the anterior ganglion-cell columns of the spinal cord are the centre of nutrition for the muscles of the trunk and limbs; hence disease in this region would cause muscular atrophy; this disease may be primary, or may be secondary to other spinal diseases, such as locomotor ataxy, sclerosis of the lateral cords, central myelitis, etc. The anterior nerve-roots, which are traversed by the muscular nerve-filaments, and even the peripheral nerves, participate more or less in the atrophy of the trophic spinal centre. The affected muscles atrophy and become pale-red or yellowish; in cases not far advanced we may find normal, fatty, and atrophied filaments in the same muscle. Sometimes, instead of fatty degeneration in the primitive filaments, it occurs in the interfibrillar connective tissue; so that a division has been made into parenchymatous and interstitial. Allied to the latter form is pseudo-muscular hypertrophy; this occurs chiefly in young males, and is due to proliferation of the interfibrillar connective tissue, with deposit of numerous fat-cells and atrophy of the muscular filaments; the deposit of fat enlarges the muscle and gives the appearance of hypertrophy. This affection comes chiefly in the lower limbs, and then in the erector spinæ muscles.

Progressive muscular atrophy is most common in males between thirty and fifty years. It is often hereditary, but is most frequently due to continued strain of certain muscles; sometimes also to cold, excesses, certain diseases, such as typhoid, diphtheria, etc., and to injuries.

Progressive muscular atrophy and bulbar paralysis differ in the location of the original disease; the former being due to changes in the motor ganglion-cells of the anterior horns, the latter to changes on the floor of the fourth ventricle.]

Symptoms and Course.—The first symptom of progressive mus-

cular atrophy is a gradually increasing feeling of weakness in certain muscles or groups of muscles, with their very evident and steadily increasing emaciation, but without pain or other trouble. The disease does not originate with equal frequency in the different muscles; but, in most cases, the muscles of one hand or one shoulder are first affected, occasionally those of the neck, rarely those of the face. The power of conduction and the size of the muscle, which diminish about equally in the course of the disease, are finally so much reduced, that the movements depending on the affected muscles are entirely lost or very imperfectly made, and, where the belly of the muscle was formerly prominent, we find a flatness or even a depression. The effect induced is most apparent in the disappearance of the ball of the thumb, depressions between the metacarpal bones, flattening of the shoulder, and prominence of the spinous processes of the vertebræ, when the muscles of the neck have atrophied. In the weak and atrophied muscles we may always see a peculiar fibrillar twitching that has no effect on the point of insertion of the muscles, and hence induces no movement of the corresponding joint; this twitching becomes more evident when we blow on the skin covering the muscle, or irritate it in any other way by exposure to cold. It is usually said that the excitability of the sensory cutaneous nerves remains normal, and only that of the sensory muscular nerves is lost (the "electro-muscular sensibility" of the electricians); but I have seen many cases where the excitability of the cutaneous nerves was much diminished, while all the other symptoms were those of progressive muscular atrophy. A very important and even pathognomonic symptom is the above-mentioned condition of the affected muscles and their motor-nerves in regard to the induced current; this current does not fail to induce contraction until the muscle is entirely atrophied; up to that time the contractions correspond to the strength of the current and the amount of muscular substance that still remains. In some cases progressive muscular atrophy is limited to certain parts of the body; in others it gradually extends to the greater part of the muscles supplied by the cerebro-spinal nerves, while it never affects the muscles of the heart, intestines, or bladder. The latter form is a terrible disease; patients affected with it preserve a good appetite and good digestion, their intellect is clear, and the mental functions are so unclouded that they can fully comprehend their lamentable loss, while they lose the use of one limb after another. In advanced cases the patients cannot walk or change their position; they must be fed too, as their arms hang useless by their sides; the changes of expression cease, the saliva runs out of the mouth, the speech is

indistinct, and the tongue cannot pass food into the pharynx, even after it has been placed in the mouth. Finally, after years of suffering, the patients die, because at last the muscles of deglutition and respiration also become atrophied, and fail to perform their functions. Some intercurrent disease of the respiratory organs, insignificant in itself perhaps, proves dangerous to the patient and hastens his end, because, owing to the paralysis of the abdominal muscles, he is unable to cough up the secretion collecting in the bronchi.

[In the last stages of some cases there is local anæsthesia; and sometimes there is contraction or faulty reaction of the pupil from paralysis of the radial fibres of the iris. Besides the atrophy of the muscles, there may be other trophic disturbances, such as atrophy of the skin, herpes in the region of the affected nerves, and affections of the joints and bones of the phalanges, carpus, etc. There may be profuse sweating, and changes in the urine, especially diminution of creatin (the product of muscular changes). Sometimes the temperature of the affected limbs is at first higher; later, it may be several degrees below normal.

Progressive muscular atrophy may be mistaken for what *Charcot* has recently described as sclerosis of the lateral cords (*sclérose latérale amyotrophique*); this sclerosis is most extensive in the cervical medulla, but may extend up into the pyramids and pons or down into the dorsal and lumbar regions. As the anterior horns of the gray substance are implicated in the disease, and undergo the same changes as in progressive muscular atrophy, the symptoms of the two diseases are mingled. But there are other symptoms which do not occur in progressive muscular atrophy; there is a true *paralysis* and a subsequent atrophy of all the muscles of the limb affected; these are followed by spastic contractions, which it is said do not occur from progressive muscular atrophy.]

Treatment.—In those forms of progressive muscular atrophy resulting from overwork and limited to certain parts of the body, the methodical use of the induced or constant current of electricity and massage sometimes arrests the progress of the disease, and even improves the nutrition of the emaciated muscles. But great patience and perseverance are requisite for the attainment of this end. All remedies, even the methodical employment of electricity, have proved inefficacious in the form of the disease which spreads from muscle to muscle.

CHAPTER IX.

PROGRESSIVE MUSCULAR PARALYSIS AS A RESULT OF HYPERTROPHY OF THE INTERSTITIAL FATTY TISSUE.

In 1869–'70 a series of very peculiar cases of progressive muscular paralysis were published, in which the volume of the paralyzed muscles was decidedly increased instead of being lessened, as it was in the myopathic paralysis described in the last chapter.

On examining the affected muscles, as was done in several cases during life by excising small pieces, they showed a changed appearance even to the naked eye. They did not have the red hue of healthy muscles, but were pale and yellowish, resembling lipoma more than muscle. Microscopic examination showed a decided preponderance of the interstitial fatty tissue over the muscular; in some places the latter was entirely displaced by the former; the muscular filaments still remaining were atrophied, pale and small, but not affected with fatty metamorphosis. Hence, in this disease there is proliferation of the interstitial fatty tissue, which probably induces simple atrophy of the muscular fibrillæ by pressure on them.

The disease has been repeatedly observed in children of the same family, and, curiously enough, only in boys. Occasionally the patients appeared to bring the predisposition to the disease into the world with them. At least, in one case observed in my clinic, whose history was published by my former assistant, Dr. *Siegmund*, in his inaugural dissertation, and in the first volume of the "Archives für klinische Medicin," and in a case described by *Griesinger*, it appeared that the patient did not learn to walk till late, and had always remained somewhat helpless.

Of course the symptoms of the disease vary with the group of muscles affected. In my patient, the disease started from the gluteal muscles. As long as these were exclusively or chiefly affected, the patient could only walk when, by aid of his arms, he had given his head and shoulders a position where their point of equilibrium fell behind the pelvis; if this position was changed, he doubled forward at the hip-joint. Now the disease affects all the muscles of his lower extremities; the patient cannot leave his bed, and can only change his position by great exertion of his arms.

I know of no cases of improvement or recovery except those of *Benedikt*, who claims to have attained positive good results in three cases by electricity. In my patients, who were treated for a long time as advised by *Benedikt* (by placing the copper pole on the lower cervical ganglion, and applying the zinc pole along the side

of the lumbar vertebræ by means of a broad metal plate), neither this treatment nor long-continued faradization of the diseased muscle had any decided effect.

ADDITIONS TO THE REVISED EDITION OF 1880.

DISEASES OF THE ORGANS OF LOCOMOTION.

1.—P. 527.

Without doubt the articular inflammations in acute rheumatism are not due to purely local causes, but evince a general morbid process. This is shown by the inflammation of numerous joints distant from each other, its disappearance from one on attacking another which was previously unaffected, the frequent implication of the heart and other internal organs, the remarkable sweating, etc. The nature of this apparently constitutional disease has not yet been solved, but there have long been two theories about it: 1. A humoral theory, according to which there is a change in the blood and fluids of the body, where from imperfect assimilation there is an excess of lactic acid; 2. A neuropathologic theory, which refers the disease to a morbid excitement of the vasomotor trophic nerve-centres; this view finds some support in the tendency to symmetrical occurrence, and in the well-known dependence of certain other joint-affections on spinal diseases.

From its frequence and severe consequences, acute articular rheumatism is one of the most important of diseases, and ranks along with consumption, pneumonia, bronchitis, typhoid fever, etc. It is rare in childhood, more frequent from the fifth to the fifteenth year, and most so between fifteen and thirty-five; after that its occurrence as a first attack is more rare. Both sexes are about equally liable. A considerable percentage shows a hereditary predisposition, especially among the younger cases. There seems to be a greater tendency to its development after certain diseases, such as scarlatina, dysentery, and in the puerperal state, after abortions, etc.

2.—P. 535.

In those cases with high fever and cerebral symptoms, we may resort to cold baths, as spoken of in the treatment of typhoid fever. In place of wrapping the joints in wadding or applying narcotic liniments, firm bandages may sometimes be applied with benefit; for this purpose we may use pasteboard moistened and moulded, or gutta-percha, placing plenty of wadding under them; after the application of such firm dressings the pain sometimes subsides very

quickly. Among remedies said to be specifics for rheumatism are propylamin, trimethalin, and more especially salicylic acid or salicylate of soda. The latter may be given in doses of 15 to 20 grs. every hour or two hours, till the pain is relieved, unless tinnitus, cerebral symptoms, or signs of collapse should require discontinuance of the medicine. The possibility of such symptoms being induced demands that the patient should be watched. Under the influence of the salicylate, the fever and local symptoms usually subside within twenty-four to forty-eight hours, the tongue cleans off, and the appetite improves; when the change has taken place, the remedy is to be carefully and gradually discontinued by decreasing the dose or lengthening the interval. The patient should be watched for at least a week. Under this remedy heart complications are said to be much less frequent. Should the joints remain swollen, we may try iodide of potash and warm baths, especially alkaline baths.

3.—P. 565.

Observers agree that the most important point in the treatment of rachitis, as well for preventing curvatures as for curing the existing disease, is proper diet. Infants should be nursed till the eruption of the first teeth, say the seventh month; then we may give in addition meat broths, egg, rare or raw shaved meat, etc. They should not be nursed over nine months or a year. When the mother cannot nurse, and a good wet nurse is not to be found, something must be substituted. About this substitute opinions are not unanimous. Some recommend cow's milk diluted and neutralized with magnesia, others *Nestle's* food, others again *Liebig's* food or condensed milk. Probably none of these is to be chosen under all circumstances; but if the child does not thrive on one, try another. *Biedert* discovered that the casein of human milk is chemically different from that of cow's milk, and claims that this is the cause of their difference, and that cow's milk cannot be made to resemble human milk simply by diluting with water and adding sugar. He recommends a mixture of one-eighth of a pint of sweet cream and three-eighths of a pint of boiled water, with the addition of 15 grs. of sugar of milk. This mixture contains the proper amounts of fat, salts, and sugar, and only one per cent. of casein, which is just about what a child's stomach will digest, according to *Biedert*. This is the proportion to begin with, but as the child grows older more cream is to be added. The yolk of egg may be mixed with water, sugar of milk, and a very little salt. Finely-ground linseed meal is said to be the chief constituent of revalenta arabica, which has some reputation as an article of nutriment.

CONSTITUTIONAL DISEASES.

The diseases to be discussed in the following sections I have termed *constitutional diseases*, in contradistinction to the *diseases of the organs*, which have been thus far described; because, from its greater comprehensiveness, this term appears to me preferable to the names dyscrasias and cachexias, or diseases of the blood, as I originally intended to call them. I shall first discuss the acute infectious diseases, then chronic infectious diseases, and, lastly, the general disturbances of nutrition, which do not depend on infection, but, at the same time, shall confine myself to those complaints which occur in Germany. In regard to exotic diseases, with which I am not personally acquainted, and for whose description I should have to rely entirely on other authors, I refer to the excellent works of *Griesinger* and *Hirsch*, where the descriptions of these diseases are as concise as they are complete.

SECTION I.

ACUTE INFECTIOUS DISEASES.

CHAPTER I.

MEASLES—MORBILLI—RUBEOLA—ROUGEOLE.

Etiology.—Measles is a purely contagious disease. There is no doubt that a person is never affected with measles without having been infected by a person with measles. This assertion has been objected to, on the ground that the *first* case of measles could not have been induced by infection, because at that time there was no measles for the patient to be infected from; and it is asserted that, if the disease developed spontaneously once, there is no reason for denying the

possibility of its doing so again. Such reasoning is idle. We know nothing about the first development of measles, and the fact, observed wherever it is possible to watch carefully the commencement and spread of the disease, that measles never occurs without a case having been brought in from somewhere else, justifies us in concluding that the same thing occurs where it is not possible to detect infection directly. It is just the same with syphilis; we know that at the present day it is only propagated by transfer; the question how the first case of syphilis originated lies outside of the circle of physical investigation. The infecting material which induces measles has not been discovered, either chemically or microscopically. We do not even know certainly whether it be organized or inorganic, and the hypothesis, that the infection is due to the transfer of small, imperceptible, vegetable organisms, is only to be preferred to other hypotheses, because it agrees better with the facts. I shall only mention a few of the reasons for this hypothesis. The period of incubation, that is, the day or week intervening between the infection and the outbreak of the malady, speaks against the infection being caused by substances which, from their chemical or physical peculiarities, are injurious to the organism. If such a substance were transferred, its injurious effects would appear at once, or in a very short time, and the infected person could not remain free from all signs of disturbance for from eight to fourteen days, and then have these suddenly break out with great severity. But if healthy persons be infected by measles patients, through microscopic organisms, the period of incubation is much more readily understood; for it is easy to suppose that these organisms are transferred in too small numbers to do harm, but that they multiply in the infected person, and immediately after this process, which is completed in a certain time (the period of incubation), their injurious influence is made manifest. A further reason for supposing that measles-poison is organized, is its reproduction in the bodies of the infected patients. For instance, in an epidemic in the Faroes observed by *Panum*, one case that was introduced infected the attendants of the patient; these infected other inhabitants of the island, and, finally, in the course of seven months, 6,000 persons, out of a population of 7,782, were attacked. If the observations of *Hallier* should prove correct, the contagious principle of measles has been recently discovered by the microscope. In the blood and sputa of measles patients, *Hallier* found microscopic cells of a fungus which grew on various substrata, but was always the same fungus, the *mucor mucdo* (*verus*) of *Fres.* It is certain that the blood, tears, and secretion of the air-passages, are vehicles for the contagion; for inoculations made with these fluids have induced measles in previously healthy persons. But, as the disease most fre

quently occurs in persons who have not come in direct contact with either the blood or secretions of measles patients, but have only been near those affected with the disease, we cannot doubt that the contagion is also contained in the emanations from the skin and lungs. From some very striking observations of *Panum*, it has been proved that this contagion in the atmosphere can, without losing its activity, be carried for miles by the body and clothes of healthy persons who have been near a patient, and who are not themselves attacked by the disease. The period of incubation lasts from ten to fourteen days. In one epidemic of measles that I carefully observed, of which Dr. *Pfeilsticker* has given some very interesting accounts in his dissertation, written under my superintendence, school-children only were at first affected, and exactly ten days later, after the latter remained home from school, a large number of smaller children, who had been infected by their brothers and sisters, were attacked. In some cases, particularly where the infected person is already suffering from some other disease, the period of incubation appears to be somewhat longer. To the question, When is measles infectious? with our present knowledge, we should say that it is most infectious while the eruption is out; that it is probably not infectious in the stage of desquamation, while numerous cases speak for its being so in the prodromal stage. The popular opinion, that measles is most catching in the desquamative stage, arises from inattention to the period of incubation. A child infected by its brother or sister breaks out with the disease while the latter is desquamating, it is true, but was infected while the exanthema was at its height, or, perhaps, even before the eruption. The probability of infection during the prodromal stage is supported by the wonderful spread of measles through schools. Great care is usually taken to keep out of the school any children who have not gotten through the desquamative stage, as well as those having any suspicious exanthema; but children with catarrh and cough are allowed to sit on the seat with well children. If the disease were only transferred by the former class, it would be quite impossible to understand why, during an epidemic of measles, a school is often entirely emptied, while the children attending another school are not affected by the disease. The predisposition to measles is very extended. Almost every one has the disease once during life; but one attack almost invariably exhausts the susceptibility to new attacks. Since measles occurs quite frequently in populated countries, most persons are attacked during childhood, and have lost the predisposition when they are grown up. It is only in this sense that measles can be called a disease of childhood. Of 196 children, under fourteen years of age, that had not had the measles, who lived in a small town near Tübin-

gen during the epidemic described by Dr. *Pfeilsticker*, 185 were taken sick; or, only eleven out of the entire number escaped. Hence, it appears that in isolated places, which are rarely visited by measles, children are not more liable to the disease than grown persons are. The above-mentioned epidemic in the Faroes, described by *Panum*, was the first that had occurred for sixty-five years in that isolated island; hence almost all the inhabitants under sixty-five years of age, who had not had the measles somewhere else, were attacked by the disease, whether they were young or old. Children under six months often escape during an epidemic of this disease. In the above-mentioned epidemic, not a child under five months was attacked; while, above six months, almost all were. Very old persons also rarely have the disease. Acute and chronic maladies, pregnancy, and the puerperal state, do not protect from measles; but, as was above said, the disorder not unfrequently makes its appearance at the end of an acute disease, during which the infection has occurred. Measles occurs in more or less extensive epidemics; the extent of the epidemic depends chiefly on the length of the interval that has elapsed since the last one, and consequently on the number of persons who have not yet been attacked. The epidemic at the Faroes furnishes an excellent example of this also. But the state of the weather appears to have a great influence on the extent of epidemics, for the greatest number and the largest epidemics occur in the winter and autumn, or in cold, damp summers. Great extent of the disease generally corresponds with great severity of the individual cases, and the most malignant cases usually occur at the height of the epidemic.

Anatomical Appearances.—The normal exanthema of measles disappears after death, and only the hæmorrhages in the tissue of the cutis, which sometimes occur, are discoverable in the dead body. The anatomical changes observed during life on the skin of a measles patient consist in an eruption of numerous roundish red spots about the size of a millet-seed, somewhat elevated, and generally having a small papule at the middle. In some places several spots unite, forming irregular semilunar patches; at other places the spots are isolated. Between the spots the skin retains its normal color; in the face, it is usually somewhat œdematous. According to *G. Simon*, the papules on the measles eruption, although chiefly located at the point where the hairs escape from the skin, are not swellings of the hair-follicles or sebaceous glands, but consist of small collections of inflammatory exudation at circumscribed points in the skin. Occasionally the eruption shows an unusual tendency to confluence (morbilli confluentes); even in such cases, however, the diffuse redness is not regular, but maintains an irregularly spotted appearance. In most of the spots which

have existed for any time, a very slight escape of blood into the cutis appears to complicate the hyperæmia, because these spots only lose their color slowly and incompletely under the pressure of the finger, and, after disappearing, they generally leave, for a time, a dirty-brown stain. In some cases the spots acquire a dark, blood-red color, from greater extravasation of blood in the cutis, and in these cases we also occasionally notice petechiæ between the spots (*morbilli petechiales*, *rubeolæ nigræ*).

Since a large number of the deaths occurring during measles result from complications with croupous laryngitis, bronchitis, or pneumonia, we often find anatomical changes due to these diseases. Where death is caused by laryngitis, it is by the croupous form. But in this secondary croup we do not find coherent pseudomembranes so often as we do in common idiopathic croup. On the contrary, the exudation usually infiltrates the upper layer of the mucous membrane, so that the inflammation approaches the diphtheritic form of inflammation of the mucous membrane. The lesion most frequently found in the bodies of children who have died of measles, is capillary bronchitis, which has sometimes induced permanent inspiratory distention of the alveoli (called acute vesicular emphysema, by most authors; see Vol. I., p. 71), sometimes collapse of the lung, and catarrhal pneumonia (Vol. I., p. 194). The blood shows no characteristic change, but, as in other infectious diseases, is poor in fibrin, fluid, and dark-colored.

Symptoms and Course.—During the *period of incubation*, there is no sign of the disease. This is followed by the first stage, *stadium prodromorum*, which rarely begins with a single chill; more frequently with repeated rigors, and is accompanied by all the symptoms of a severe catarrh of the conjunctiva and air-passages, and the actual disease can only be rightly interpreted from the existing epidemic. Unless it be known that there is measles in the neighborhood, the most experienced physician can hardly recognize in the existing severe catarrh the prodromal stage of measles. Increased frequence of pulse, heightened temperature, constitutional disturbance, pain in the head and limbs, dyspepsia, nausea, vomiting, disturbed sleep, and, in excitable children, delirium, occur also during simple catarrh, from catching cold. The local symptoms are generally very decided; the burning, reddened eyes shun the light, and are filled with tears, there is pain in the forehead, the nose is stopped, and discharges a copious, limpid, salty secretion; attacks of sneezing occur at short intervals, and often continue for hours; there is, sometimes, epistaxis, the voice is husky, the painful cough is hoarse and barking; at night, the attendants are often frightened by the symptoms of pseudo-croup, previously described (Vol. I., p. 6). The catarrhal affection appears generally to begin in

the nose, and to extend upward to the mucous membrane of the frontal sinus and conjunctiva, and downward to that of the larynx and trachea. The stadium prodromorum usually lasts about three days, while the above symptoms are of variable intensity; but in some cases it lasts a week or longer, and in others the symptoms are very slight, and readily overlooked. Even in malignant epidemics, the fever preceding the eruption is rarely severe enough to threaten life, as it is in scarlet fever. Nor are the local symptoms, severe as they often are, and terrifying as they seem to the friends (especially the croupy cough and nocturnal dyspnœa), usually dangerous. True croup rarely occurs at this time. *Pfeilsticker's* observations of the course of the fever during the prodromal stage showed that the temperature was highest the first day, and subsequently sank till the day of the eruption. *Ziemssen* and *Rehn* also mention cases where, after the bodily temperature had attained a considerable height the first day, it became perfectly normal the following days, and remained so till the eruption appeared, when it rapidly increased. During the prodromal stage, *Rehn* observed an eruption of pale-red, rather undefined spots on the mucous membrane of the cheeks, gums, lips, and fauces; and he explains the increase of temperature at the commencement as being the eruptive fever of this exanthema.

The second stage of measles, the *stadium eruptionis*, commences with an exacerbation of the fever; the pulse becomes more frequent, the bodily temperature rises to the highest point; in some cases there are convulsions. The above-described eruption appears first in the face, especially about the mouth and eyes; it soon spreads to the neck and breast; even in twenty-four hours it usually reaches the feet, so that the whole body is covered. At this time the perspiration of the patient has a peculiar odor, which strongly reminds me of a freshly-picked goose. In rare cases, which generally show some other anomaly also, the eruption does not spread from the face to the extremities, but makes its first appearance on the arms or legs, and afterward elsewhere. Still more rarely, the exanthema is limited to certain regions of the body, or, at least, on the rest of the body it is very indistinct. These cases resemble the *morbilli sine exanthemate*, which unmistakably results from infection with measles poison, but from first to last runs the course of a very severe catarrh, without the appearance of any eruption. Lastly, cases occur where the eruption comes out so slowly, that the stadium eruptionis is not completed in twenty-four or thirty-six hours, but continues to the third or fourth day. In these cases, the last spots frequently do not appear till the first ones are fading away. During the eruptive stage, the constitutional disturbance and catarrhal symptoms usually increase, and attain their

height as the exanthema reaches its full extent. The *stadium florescentiæ* cannot be distinctly distinguished from the eruptive stage, for in most cases the exanthema is just at its height as the eruption is completed, and it generally begins to disappear in twenty-four hours. The fever, also, which usually attains its height at the completion of the eruption, rapidly moderates, and sometimes disappears in the stadium florescentiæ, so that the parents have great difficulty to keep in bed the little patients, who are often still covered with red spots, but are already full of spirits. The catarrhal symptoms continue, it is true, but they are much milder; the photophobia is less, the secretion from the nose is more scanty and thick, sneezing is rarer, the voice less rough, the cough looser, and older children, who do not swallow the sputa, cough up more or less muco-purulent masses (sputa cocta). On the third or fourth day after their occurrence, the spots, especially those of the first crop, are ordinarily much paler, or have entirely disappeared, commonly leaving a bright-yellow discoloration on the skin for a time.

In favorable cases, the fourth stage, *stadium desquamationis*, usually succeeds the stadium florescentia the eighth or ninth day of the disease. At this time the spots have entirely disappeared, and their former seat is covered by a bran-like desquamation of the epidermis. If the detached epidermic scales be softened, and macerated by constant perspiration, the desquamation is not so evident as if the skin be dry; hence, it is less perceptible on the parts covered by the bedclothes than on the face, neck, and hands. In the desquamative stage, the fever has almost always disappeared. The catarrh also passes off gradually, and about the fourteenth day of the disease, or somewhat later, as the desquamation ends, the measles terminates.

In many cases the disease runs its course without much deviation from the description above given. These cases, where there is no material deviation from the "normal" course, and where the different stages are not accompanied by any peculiar symptoms, are usually called *morbilli vulgares*, *simplices*, or *erethici*.

In other cases, usually called *inflammatory* or *synochal* measles, the exanthema appears with very violent symptoms; the spots, which are usually close together, and often confluent, do not begin to lose color in twenty-four hours, but at this time grow darker, and remain visible on the skin for five or six days. Occasionally the exanthema assumes a violet or bluish color, and does not disappear under the pressure of the finger; this depends on partial rupture of the overfilled capillaries, and may be regarded as analogous to the hæmorrhages occurring in the inflammatory disturbances of nutrition. The correctness of this view is supported by the fact that *this* form of hæmorrhagic measles usually runs a favorable course, and is not at all

complicated with the signs of dissolution of the blood. The fever symptoms accompanying so-called inflammatory or synochal measles resemble those observed in the course of acute inflammations. The action of the heart is increased, the carotids pulsate strongly, the pulse is strong, full, and more frequent, but not excessively so; the bodily temperature does not much exceed 104°. Just as in the case of the exanthema, the affection of the mucous membranes is characterized by longer duration, greater intensity and extent, in synochal measles. As long as the exanthema is at its height, the photophobia, nasal catarrh, and cough remain the same. It is this form of measles also which is chiefly accompanied by croupous instead of catarrhal laryngitis, in which the inflammation of the air-passages often extends to the alveoli of the lungs, in which also the gastric and intestinal mucous membrane is often affected with catarrh (gastric measles). If a hoarse, barking cough and the dyspnœa characteristic of croupous laryngitis occur in the stage of efflorescence, the state of the patient is by no means so free from danger as if these symptoms accompany the prodromal stage; but in such cases the disease readily takes a bad turn, the dyspnœa increases, the breathing is impeded, and, as the skin becomes congested, the exanthema subsides, or, if it has been accompanied by hæmorrhage in the cutis, blue spots remain, which do not disappear on pressure. In regard to the modification of the symptoms induced by an extension of the catarrh to the finer bronchial tubes, we must refer to the first volume, where we have fully described the symptoms of capillary bronchitis and the dangers accompanying it, particularly during childhood. The description of collapse of the lung and catarrhal pneumonia in the first volume also gives the symptoms from which we may determine that these complications exist. Indeed, it is to the numerous observations of collapse of the lung and catarrhal pneumonia, afforded by extensive epidemics of measles, that we owe our accurate knowledge of the pathological anatomy and symptomatology of these diseases. Far more rarely than catarrhal pneumonia we have croupous pneumonia during synochal measles; it is almost always limited to one side, while catarrhal pneumonia usually affects both sides; the fever accompanying the latter is ordinarily insidious, and disappears gradually, while that with the former runs a cyclical course and defervesces suddenly. The course of the fever due to the measles is decidedly modified by the above complications. This is especially true in the case of the pneumonia. While, in uncomplicated measles, the fever, which has reached its acme at the height of the eruption, subsides steadily, and often rapidly, it may attain its acme much later when there is a complication with catarrhal or croupous pneumonia. If measles be accompanied by severe disease of

the respiratory organs, and the consequent difficulty of breathing or fever induce collapse, the eruption becomes pale, and may entirely disappear in a short time. This symptom is often falsely interpreted, as the disappearance of the eruption is regarded as the cause, not the result, of the distress and of the bad symptoms in the respiratory apparatus.

A third class of measles described is the asthenic, typhous, or septic. In this very malignant form of the disease, the danger lies, not in the extension of the catarrh to the bronchi, or in severe complications, but in the pernicious influence of the measles poison on the entire organism. Measles resembles most acute infectious diseases in regard to the great difference in the constitutional disturbance induced by the infection during different epidemics. A physician who has only observed epidemics of erethitic or synochal measles, such as have exclusively occurred in Germany during the past ten years, may readily suppose that measles poison has little constitutional effect. But this view will soon be altered the first time he sees a case of asthenic septic measles. Even in the prodromal stage, the pulse, which is at first full and strong, may become small, weak, and very frequent; the patient very much depressed, the intellect cloudy, the tongue dry and crusted, and patients may die of the increasing prostration, which is occasionally interrupted by eclamptic spasms, even before the appearance of the eruption. In other cases, these typhoid symptoms, which are often accompanied by abundant epistaxis, do not begin, or do not become dangerous, till the outbreak of the eruption. The exanthema, which is usually irregular, is sometimes pale red; sometimes bluish-violet, as a result of coincident hæmorrhage into the skin; occasionally there are petechiæ between the spots, or bluish spots remain after the exanthema has quickly disappeared. When the pulse has become very small, and cannot be counted—the extremities cool, while the body is hot—most patients die in a soporose state, with or without general convulsions. It has not yet been accurately ascertained whether the adynamia and paralysis occurring during measles, inducing the so-called asthenic, typhous, or septic form of the disease, be a direct result of the blood-poisoning, or if they be due to the excessive increase of bodily temperature induced by the infection. The latter view is apparently supported by the fact, that in diseases not induced by infection, as soon as the bodily temperature rises above a certain point, the pulse becomes small and weak, and the same nervous or typhoid symptoms appear; as well as the second fact, that remedies which lower the bodily temperature have a decidedly favorable effect on these symptoms.

The cough continuing during the desquamative stage forms a con-

necting link with the sequelæ of measles; for it often remains for weeks or months after the desquamation is over, and grows worse from the most trifling causes. In many cases this cough appears to depend on simple bronchial catarrh, and its obstinacy and occasional exacerbations seem due to increased vulnerability of the bronchial mucous membrane, or to a sensitiveness of the skin remaining after measles; but, in other cases, the cough is more serious, and depends on severe disease of the lungs. It is well proved that measles often induces pulmonary consumption, and, consequently, that after an extensive epidemic of measles, the rate of mortality among children is usually increased for a few years. But it appears to me very doubtful whether the chronic pulmonary tuberculosis, often induced by measles, is always or chiefly due to a deposit of tubercles in the lungs during measles, or to softening of tubercles already existing there. Most cases of phthisis pulmonum in children are not due to deposit and softening of miliary tubercles, but to caseous transformation and disintegration of the products of lobular pneumonia, which is generally also accompanied by caseous degeneration of the bronchial glands. But as lobular pneumonia is one of the most common complications of measles, it appears very probable that the frequent occurrence of pulmonary consumption, as a sequel of measles, is due to further metamorphosis of the products of inflammation, which have not yet been resolved. But this does not mean that true tuberculosis of the lungs *never* occurs as a sequel of measles (for I have several times seen acute miliary tuberculosis directly follow measles), but only that *most* cases of pulmonary consumption after measles depend on chronic destructive pneumonia. Besides chronic bronchial catarrh and chronic pulmonary consumption, the whole series of scrofulous diseases, especially ophthalmia, otorrhœa, chronic rhinitis, swelling of the lymphatic glands, chronic inflammation of the periosteum and joints, may be mentioned as sequelæ of measles. At least, we very frequently see children, who had never before suffered from scrofula, troubled for years with various forms of these chronic inflammations after recovering from an attack of measles. Among the sequelæ of measles, which, fortunately, are rare, we may mention the mortification of the cheeks or vulva, noma, and the diphtheritis of the oral mucous membrane, stomacace, which occur even during desquamation.

Treatment.—The only efficient prophylaxis is a strict isolation of healthy persons, who have not yet had the measles, from those in whom the disease has broken out, as well as from those that are suspected of being in the prodromal stage. To protect children from the affection, it is necessary to keep them out of school; and it is still better, if possible, to send them away from the place where an epi

demic is raging. This plan will probably only be adopted in those rare epidemics that are characterized by their malignancy and fatality; in mild epidemics this will not be done, because we know that sooner or later almost every person has the measles. As inoculated measles is not milder than other cases, and as malignant epidemics are rare, inoculation during mild epidemics has not proved popular.

If measles has once broken out in a person, we must remember that we have no remedy for cutting short the attack; also that the medicines usually given for the different symptoms are not only uncertain, but often act injuriously on the course of the disease; lastly, that the great majority of cases end in recovery without any medical treatment. If we remain convinced on this point, we shall abstain from superfluous and dangerous medication. To most of the patients we shall probably give no medicine, but only order proper regimen, and shall only act energetically when actual danger threatens. All measles patients should be kept in an even and moderate temperature. The physician should insist that the temperature of the room be regulated by the thermometer, not by the feelings of the nurse, and kept at about 60° to 65°. And the chamber must be aired every day, while a thin cloth is thrown over the head and face of the patient, and his bed is protected from draughts by a screen. The old custom of leaving a measles patient at least fourteen days without washing him, or changing his linen, has very justly been given up, since it has been proved that, notwithstanding the dirt, the exanthema sometimes disappears, but in most cases does not do so, in spite of careful daily ablutions. But this excess of care is no worse than neglect of attention when washing or changing the linen of the patient. Both of these operations must be done quickly, and without unnecessary exposure. Lukewarm water is preferable to either cold or hot. The body and bed-linen should not be taken from the clothes-press and placed directly on the patient, but should be first warmed, and aired, or, still better, worn for a night by some well person. The chamber should be darkened in proportion to the degree of conjunctivitis and photophobia. If we make the room too dark, by thick, green curtains, we render the eye-trouble worse, for the patients are dazzled every time the door is opened, and light enters the room. In the prodromal as well as in the eruptive stage, we should order absolute diet, water-soup, bread, and, if there be constipation, stewed fruit. If the fever subsides during the stage of efflorescence, we may give meat broth and milk, and, in the desquamative stage, let the patient return gradually to his customary diet. Simple cold water, that has stood in the room for a while, should be freely given, as it is never injurious, and does not even render any of the symptoms worse temporarily. On the other

hand, it is cruel and injurious to withhold from the patient the only thing that will quench his thirst, as long as he is feverish and thirsty, and in place of it to make him drink warm tea or water. Sweet drinks, which generally soon become distasteful to the patient, and do not alleviate the cough, as they are expected to, are superfluous. The time that the patients are to pass in bed, and in their chamber, should not be measured in the customary way by days and weeks, but we should insist on his remaining in bed as long as there is any indication of fever, while the desquamation is going on, and the cough is severe, and he should keep his chamber as long as there is any trace left of the measles-catarrh, whether the customary fourteen or forty-two days have elapsed or not. Even after the patient has fully recovered, the physician should keep a watchful eye on him for months, and should particularly attend to any cough, no matter how slight and insignificant it may appear. Among the accidents that demand active treatment, during measles, most authors place the "striking in of the eruption" in the first rank, and consider its "restoration" as the most important point in treatment. We do not hesitate to say that it is just as unscientific as it is dangerous to carry out this indication; it is dangerous because it readily induces rules which may have an injurious effect on the course of the disease. As above shown, the so-called disappearance of the eruption is not to be regarded as the cause, but as the result of a bad turn of the disease, and is due to the general collapse of the patient, in which the skin participates; but, as we have also shown above, this bad turn of the disease usually depends on the appearance of some complication, especially pneumonia; if this fact be not borne in mind, if the patient be rubbed with irritating tinctures and liniments, placed in a hot bath containing mustard or caustic potash, or wrapped up in blankets wet with decoction of mustard, "to bring the eruption out again," we shall often do much harm, even if we succeed in our object, because these procedures do not generally act advantageously on the pneumonia and other complications, while they increase the fever. Among the symptoms of the prodromal stage, the attacks of hoarseness, aphonia, and severe dyspnœa, which occur, particularly during sleep, require the same treatment as when they occur in the course of genuine laryngeal catarrh, such as emetics, hot applications to the throat, and plenty of warm drinks. If the cough be uncommonly severe and persistent, we may give adults five or ten grains of Dover's powder at night, and for children, where opium is dangerous, especially during fever, we may prescribe small doses of lactucarium, or a weak infusion of ipecac. (gr. vj to ℥ iv water, with syrup ℥ ss). If, during the stage of eruption and efflorescence, the fever show a synochal character, there is no reason why we should not give

nitrate of soda, although it will very rarely prevent any inflammatory complications. Great care must be exercised in the employment of tartar emetic, for violent vomiting and purging are injurious to measles patients, and readily induce sudden collapse, even during so-called inflammatory measles. Laryngitis and pneumonia complicating measles are to be treated in the same way as when they occur primarily, although the prognosis is not so good; the same is true of other complications. In asthenic, typhous, and septic measles, quinine, mineral acids, and stimulants, are most frequently given. I have not seen any malignant measles recently; but should it prove that they induce a very high bodily temperature, from my experience in analogous forms of other infectious diseases, I should not hesitate in such cases also to wrap the patients up in cold, wet cloths, at short intervals, and to give them large doses of quinine.

CHAPTER II.

SCARLET FEVER—SCARLATINA.

Etiology.—Scarlatina is an infectious disease, as is shown by a few cases of successful inoculation, and by many well-known instances where the disease was carried from one place to another by scarlatina patients. It is less clearly proved that contagion is the only mode of propagating the disease, and that scarlatina never develops spontaneously. At least, epidemics have occurred in places where its extension could be readily observed without its being detected, or even being probable that the disease had been imported. We know no more about scarlatina poison than we do about that of measles. The infection of persons who have been near scarlatina cases, without being immediately in contact with them, appears to show that the poison is contained in the exhalations of the patient, and is mingled with the atmosphere about them. Well-proved facts also render it probable that the contagion may be carried by persons who are not themselves affected by the disease. Past experience has not yet taught us whether the blood and secretions are also means of conveyance. The period of incubation appears to be shorter than in measles, and to last only eight or nine days. But, from the difficulty of deciding the time when the infection terminates, this point is not exactly determined; and, for the same reason, we cannot accurately answer the question, In what stage is scarlatina infectious? The predisposition to scarlatina is far less common than that to measles; there are not a few persons who never have it. One attack almost unexceptionally removes the liability to another. Nursing children often escape during an epidemic; while

those over two years old are most susceptible to the disease; but even adults, who have not had scarlatina during childhood, are often attacked by it, and cases occur even in old age. In very large cities it seems never to disappear entirely; we do not know why the disease occasionally spreads, and why, after occurring sporadically for a year, it should suddenly break out into an epidemic. Nor do we know the causes of the differences between epidemics in regard to the mildness or malignancy of the attacks. Epidemics of scarlatina occur chiefly, but not exclusively, in autumn and spring; they succeed each other at longer or shorter intervals, and are of very variable duration.[1]

Anatomical Appearances.—The anatomical changes in the skin during life are those of a very extensive erythematous inflammation, consisting of a very intense hyperæmia and an inflammatory œdema of the superficial layers of the cuticle. The redness of the skin caused by the hyperæmia begins as numerous small points, close together, which soon unite and form an even redness (*scarlatina lævigata*). Far more rarely the redness is limited to a few spots of variable size and irregular shape, or the surface is pale red, with some darker red spots over it (*scarlatina variegata*). More rarely than in measles, collections of inflammatory exudations at circumscribed spots form small papules (*scarlatina papulosa*); on the other hand, exudation effused on the surface frequently elevates the epidermis to numerous small vesicles (*scarlatina miliaris seu vesicularis*), or to larger vesicles filled with limpid or cloudy-yellow liquid (*scarlatina pemphigoidea seu pustulosa*) In malignant cases, the hyperæmia of the skin is occasionally accompanied by more or less extensive hæmorrhages, causing petechiæ and extensive ecchymoses. If death occurs at the height of the scarlatina, the skin of the cadaver often appears thickened and hardened by infiltration, and we frequently find dried vesicles and dark-colored petechiæ on it, that have not disappeared after death as the hyperæmia has done.

Inflammation of the *pharyngeal mucous membrane* is induced by scarlatina poison just as often as inflammation of the cutis is. The most frequent form of this pharyngitis is the catarrhal, in which the mucous membrane of the palate, tonsils, and pharynx, is dark red, swollen, at first dry, but subsequently covered with quantities of mucus. In malignant epidemics, scarlatina is not unfrequently localized in the pharyngeal mucous membrane as a diphtheritic inflammation; in such cases, the fauces and pharynx are at first covered with gray plaques, which cannot be wiped off from the mucous membrane, because they are due to infiltration of its tissue with fibrinous exudation. After lasting for a short time, this infiltration causes necrosis of the patches of membrane, which are thrown off as discolored, badly-smelling sloughs; and irregular losses of substance, covered with disinte

grated tissue and dirty sanies, are left. In many cases this process advances through the fauces to the nares, inducing the notoriously malignant scarlatina coryza. The diphtheritic inflammation also occasionally extends to the mouth and lips, and particularly to the angle of the mouth, while it rarely attacks the larynx. Lastly, in some cases, scarlatinous pharyngitis runs its course as a parenchymatous angina, inducing great swelling, and, after a time, suppuration of the tonsils.

Combined with the malignant pharyngitis, or even independent of it, we often have inflammations of the parotids and lymphatic glands and of the connective tissue of the neck; these rarely end in resolution, but generally after a hard swelling of the inflamed parts has lasted for a variable period, they terminate in suppuration or diffuse necrosis.

Scarlatina is localized in the *kidneys* just as often as it is in the skin or mucous membrane of the pharynx. The usual form of this localization is an intense hyperæmia and catarrh (throwing off of the epithelium) by the uriniferous tubules; but, in many cases, and in some epidemics in almost all the cases, the scarlatina infection induces croupous inflammation of the uriniferous tubules instead of simple hyperæmia. We should not consider either diphtheritic angina or morbus Brightii among the complications of scarlatina; or else we shall have to regard the inflammation of the skin, catarrhal pharyngitis and hyperæmia of the kidneys as complications and not symptoms of scarlatina.

Perhaps the same thing is true of inflammation of the joints, pleura, pericardium, and also with that of the ear, cornea, and other rarer disturbances of nutrition observed during the course of this disease; at all events, there is no proof that, in cases where these inflammations have occurred in scarlatina patients, there has been any second cause, besides the scarlatina infection, acting on the patient to excite these diseases as *complications*. It is, at least, fully as probable that the infection, which generally only induces palpable changes in the skin, throat, and kidneys, decidedly modifies the nutrition of the entire body, and, under some unknown circumstances, excites severe, and consequently perceptible, changes in the organs above mentioned.

Lastly, we may mention that, in the bodies of those who have died of scarlatina, the blood is generally poor in fibrin, thin and dark, and occasionally the spleen and intestinal glands are tumefied.

Symptoms and Course.—It is customary to give the name simple, normal, or benign scarlet fever to those cases where the fever maintains a synochal character, and where the perceptible localizations of the disease are limited to the exanthema, severe catarrhal angina,

and simple hyperæmia of the kidneys. We shall first describe the symptoms and course of this simple form (which is, however, quite a serious disease), and subsequently shall speak briefly of the modifications caused partly by the asthenic character of the fever, probably from the great increase of bodily temperature, partly by the extent and variety of the local affections.

During the stage of incubation, some patients complain of weariness and depression, and an undefined feeling of sickness; but most of them feel quite well.

The prodromal stage begins with repeated rigors, more rarely with a single chill. This is followed by a feeling of burning heat, nausea, or actual vomiting, severe headache, feeling of great relaxation, pain in the limbs, and the series of symptoms that accompanies almost any severe fever. Even at this time the pulse is often 120 to 130 beats a minute, and the bodily temperature occasionally rises to 104° to 105°, or higher. Such an increase of the pulse and rise of temperature are not often observed at the commencement of other diseases, even in inflammation of important organs, hence these symptoms alone should excite the suspicion that we are dealing with the first stage of an infectious disease. And, since, along with the fever, the subjective and objective symptoms of pharyngitis usually occur, we may generally decide with great probability, even at this time, that the case is one of commencing scarlatina, and not of commencing measles, small-pox, or other infectious disease. The patients complain of a feeling of dryness and heat in the throat, and of pain, which is increased by swallowing. On examining the throat, we find the mucous membrane of the tonsils and soft palate dark red and swollen. And in some cases the tongue is already very red at the edges. Occasionally, the prodromal stage only lasts a few hours, or the eruption may occur almost at the same time with the fever, so that there is no real prodromal stage; but in most cases this stage lasts one or two days, and rarely longer. The intensity as well as the duration of the prodromal stage varies in different cases; this is partly due to the individuality of the patient, partly to unknown causes. Some patients are very much excited or delirious; others lie benumbed and apathetic; children not unfrequently have convulsions, just as they do in other febrile diseases. Other patients are less severely affected by the prodromal stage of scarlatina, and have no serious symptoms.

The *stadium eruptionis* almost always begins with an exacerbation of the fever. The symptoms accompanying the fever, also the headache, weakness, excitement, or apathy, increase, and it is at this time that convulsions most frequently occur in children. The eruption does not come first on the face, as that of measles does, but begins on the

neck, and thence extends over the body. Even in twenty-four or thirty-six hours the whole surface is usually covered with a scarlet color. In the face, the cheeks only are generally red; hence the exanthema is less readily recognized here than elsewhere. The deepest redness is usually on the neck, outside of the limbs, joints, hands, and feet. The exhalations of the patient are said to smell like mouldy cheese, or caged wild beasts in menageries. When the eruption appears, the throat troubles increase, the fauces become more intensely red, the tongue is of a dark raspberry redness, not only at the edges, but also on its dorsum, from which the previous coating is thrown off; the swollen and erect papillæ give the surface of the tongue a rough appearance (cat's tongue). During the stage of eruption there are variations from the above symptoms, which have no material effect on the course of the disease. For instance, in some cases the exanthema spreads over the surface with uncommon rapidity; sometimes the redness is brighter, at others much darker; in other cases the exanthema presents the characters of scarlatina miliaris. In the same way the throat troubles are sometimes very severe, again very slight; they are rarely accompanied by catarrh of the larynx, trachea, and bronchi.

In the *stadium florescentiæ*, which usually lasts four or five days, the fever at first increases, reaching its height about the second day. At the same time the eruption is in its bloom, and the throat trouble has reached its height. The urine contains quantities of detached epithelium, and often, traces of albumen; the general health of the patient is also most affected at this time. Then all the symptoms usually begin to decrease, the frequency of the pulse and the temperature go down, the exanthema fades, the difficulty of swallowing grows less, the general health improves. In this stage also, besides the material deviations due to increase of the fever, or extensive and unusual local affections, different cases do not agree in all respects; occasionally, the exanthema remains out longer, or it fades and disappears earlier; the same is true of the changes in the pharyngeal mucous membrane, and of the fever symptoms.

The *stadium desquamationis* usually begins on the fifth day after the appearance of the eruption. While the eruption continues to some extent in the extremities, especially about the joints, it disappears about the neck, and here we first notice that the skin is rough and dry, the epidermis is raised, and falls off in small shreds. A few days later the redness usually disappears from the extremities also, and desquamation begins. This does not occur in the extremities as it does in the neck, with a detachment of small scales, but large flakes come off, particularly from the hands, and they are not unfrequently peeled off by the patient. During the stage of desquamation, which

lasts from eight to fourteen days, the last traces of fever and angina disappear, and, when the disease runs a favorable course, it ends in perfect recovery during the third or fourth week.

Scarlatina sine exanthemata and scarlatina sine angina belong to the simple, or, at all events, to the benign cases. The former is only to be distinguished from simple angina by its occurrence during an epidemic of scarlatina, by the high fever, by the great constitutional disturbance, and by a marked prodromal stage. In scarlatina sine angina, the fever symptoms and the exanthema are characteristic; but localization of the scarlatina process on the pharyngeal mucous membrane either cannot be found, or is limited to slight pain in swallowing, or to faint redness of the fauces We should be very careful about diagnosing scarlatina sine angina, particularly if it cannot be proved that the patient has been near scarlet-fever patients; from the great similarity of the eruption, scarlatina sine angina can occasionally only be diagnosed from some forms of roseola, especially roseola ab ingestis, by considering the causes. Like measles, scarlatina may prove very injurious, without any dangerous local symptoms; the patients may even die of the disease before the customary local symptoms have appeared on the skin or pharyngeal mucous membrane. In such cases the patients die of paralysis of the heart, which is preceded by the symptoms of excessive adynamia. Here, as in the case of malignant measles, we shall not attempt to say whether the alteration of the blood has a directly paralyzing effect on the nervous system, and especially on the nerves of the heart, or whether the injurious effect be due to the high grade of the fever, that is, to the excessive increase of bodily temperature. But since, even in simple benign scarlatina, the temperature is usually very high, and since, as we have before shown, a still further increase destroys life, as is proved by physiological and pathological experiments, we regard it as very probable that the excessive increase of bodily temperature in malignant, asthenic, or typhoid scarlatina, induces the paralysis. The symptoms of asthenic or typhous scarlatina closely resemble those of asthenic or typhous measles and the same forms of other diseases, especially infectious diseases. Even in the prodromal stage, the patients are very much depressed; they lie in a state of apathy, cannot collect their thoughts, scarcely answer any questions, and finally become perfectly comatose The pupils are usually dilated. Slight twitchings of the muscles, and, in children, general convulsions, not unfrequently occur; the tongue becomes dry, the pulse small, and scarcely to be counted; the body is often burning hot, while the extremities are cool. Even before the eruption appears, the patient may die with the symptoms of excessive collapse, and œdema of the lungs. The scarlatinous sore throat, which

from the severe constitutional disturbance, is often overlooked in such cases, is frequently of diphtheritic nature. If the patient do not die during the prodromal stage, the eruption usually appears slowly and irregularly, is pale or livid, only remains on the skin for a short time, and is often accompanied by petechiæ, which remain after the disappearance of the exanthema. The appearance of the eruption does not generally induce any amelioration of the patient's symptoms; on the contrary, the high fever continues, the pulse becomes weaker, the prostration greater. Diarrhœa and meteorism often occur, and the tongue and gums are covered with dry sordes. Most of the patients die in this stage of the disease, and of the few who reach the stage of desquamation the greater part die of the sequelæ.

Cases, where angina maligna, infiltration, and suppuration of the lymphatic glands, or other disturbances of nutrition, of themselves dangerous, occur, may run just as malignant a course as those where general paralysis is induced, directly or indirectly, by the excessive fever; we can only point out the various forms of the disease due to malignant local affections, because any exhaustive description of them would be impossible within the limits proposed for this work. Angina maligna not unfrequently occurs during an apparently benign, or so-called normal scarlatina, and in its first stages is not by any means universally accompanied by dangerous symptoms; the difficulty of swallowing only attains a high grade when, as happens in many cases, there is at the same time a parenchymatous inflammation of the tonsils, which often renders swallowing impossible. The participation of the nasal cavities in the diphtheritic inflammation of the fauces is so constant that the occurrence of a coryza, with a discharge of secretion which is apparently bland and has no peculiar odor, is a very suspicious and usually threatening symptom. This coryza rarely accompanies the catarrhal form of scarlatinous angina; hence we should dispel the illusions of the parents, who consider this discharge from the nose as favorable, and, when they see it, expect their children to escape any cerebral trouble. The only certain point for the diagnosis of diphtheritic angina, in its first stage, is the examination of the fauces, which shows the above-described dirty-white, firmly-adherent plaques on the dark-red mucous membrane. But, even in a few days, the symptoms change, and the disease appears very dangerous; it is true the exanthema offers no peculiar symptoms, but in the vicinity of the patient we may perceive a fetid odor, which comes from his mouth and nose. The sloughs, changed to dirty tags, adhere to the fauces, or, after they have been detached, unhealthy-looking ulcers form. A yellowish, badly-smelling secretion flows from the nostrils over the cheeks, and corrodes the skin with which it comes in contact; the cervical glands,

also, are much swollen, and form unsightly hard lumps at the sides of the neck. The patient usually lies with the head thrown back, in a half comatose or perfectly unconscious state; the pulse, which was previously full, becomes small and beats 140 to 160 times a minute; the bodily temperature rises to 104° or even higher. In such cases, just as in measles complicated with lobular pneumonia, the fever induced by the scarlatina infection becomes excessive from the accompanying pharyngeal inflammation, and the bodily temperature is elevated to a point where adynamia and paralysis always occur. Laryngitis, complicating this form of angina, occasionally hastens the fatal result; it is characterized by hoarseness and dyspnœa rather than by croupy cough. If the patient lives through the stage of efflorescence, desquamation often takes place normally; but, in the most favorable cases it lasts a long time, till the ulcers in the neck have healed, and till the discharge from the nose has ceased. Quite often the inflammation extends through the Eustachian tubes to the tympanum, resulting in otitis interna, which leads to perforation of the drum, and often also to caries of the petrous bone. Hence, after scarlatina, many patients have otorrhœa for years, and suffer from deafness for the remainder of life. If the diphtheritic process has extended to the mouth and lips, the resulting ulcers, especially those at the angles of the mouth, heal very slowly. During the stage of desquamation and convalescence, the patients are liable to great danger from inflammatory infiltration of the lymphatic glands and subcutaneous connective tissue of the neck. These rarely end in resolution; and even suppuration results slowly, and is accompanied by fever, which wears out the patient. I have seen patients die from suppuration of these enlarged glands, even six or eight weeks after recovery from the scarlatina. Inflammatory infiltration of the cervical glands may also develop without angina maligna, and in such cases also it induces increase of the fever and consequent "typhoid symptoms." It is more probable that the brain symptoms are due to the high fever accompanying this infiltration of the glands than that they are caused by pressure on the vessels supplying blood to the brain. Parotitis, which sometimes occurs during the stage of desquamation, just as it does in the course of typhus and cholera, must not be confounded with the above inflammations and suppuration of the lymphatic glands. It is chiefly in this stage, too, that we see inflammations of the synovial membranes, pleura, and pericardium.

We have mentioned *croupous nephritis* as a frequent and important localization of the blood-poisoning in scarlatina. The fact that scarlatina dropsy, which generally depends on this localization, usually makes its appearance during this stage, has given rise to the still preva-

lent belief that the dropsy and renal disease are due to cold, to which the patient has been exposed during the height of the exanthema or during the desquamative stage. The fact that albuminuria and dropsy are very rarely seen in some epidemics, while they occur in almost every case in others, sufficiently proves the error of this hypothesis, although we cannot explain the causes of the difference in frequency of nephritis, angina maligna, and other local affections in different epidemics. We have already (Vol. II., p. 12) fully described the symptoms and course of croupous nephritis, and we can now refer to that description the more readily, because most of the cases of croupous nephritis, which formed the basis of that description, occurred in children in the desquamative stage of scarlatina. While part of the patients affected with hydrops scarlatinosum, which is a symptom of acute inflammation of the kidney, recover and another part die with the symptoms of uræmic intoxication, and still more of pneumonia, pleurisy, pericarditis, etc., the second form of scarlatinous dropsy, not accompanied with albuminuria, is a sequel of scarlatina as free from danger as it is inexplicable. It generally develops gradually, may become very extensive, is not limited to the subcutaneous connective tissue, and rarely extends to the serous cavities. In some cases of very extensive scarlatinous dropsy without albuminuria, recovery takes place in a remarkably short time, as I know from my own observation.

Treatment.—Prophylaxis requires the isolation of healthy persons from those affected with the disease and from those who have intercourse with such patients. This is the only rule that promises any good results; hence it should be urgently recommended during malignant epidemics. Belladonna (extract belladonnæ gr. iij, aq. dest. ℥ j; give daily twice as many drops are there are years in the patient's age) is regarded as a prophylactic by some physicians as well as by the homœopaths; but experience has shown that, even when continued for weeks, this remedy affords no protection against scarlatina. The same is true of all other medicines, either internal or external, that have been recommended as prophylactics. The same rules apply to the treatment of scarlatina as we gave for that of measles. Before asking ourselves what we shall prescribe, we should decide whether it is necessary or even admissible to interfere actively with the regular course of the disease. We should keep the sick-chamber at an even temperature, not exceeding 55° to 65°, not allow the patient to be covered with heavy bedclothes, freshen the air of the room occasionally by opening the window, and have the patient carefully washed daily. The best drink is pure cold water or lemonade; as nourishment, we may at first give water-soup, bread, stewed fruit, etc.; later, we may give meat-broths, milk, etc. If there be constipation, we may order

an enema of tepid water, avoiding active purges, except in case of necessity. It is an old custom to keep scarlatina patients in bed till the stage of desquamation is over, and it is well to keep up this custom, undeterred by the fact that patients neglecting this rule do not, by any means, always suffer for so doing. Even after desquamation is completed, the patient should be protected from cold, and in winter should be kept in his room for at least a fortnight. During this time he should take several baths in lukewarm water. In simple, mild cases of scarlatina this expectant plan is sufficient, and should be preferred to any medicinal, hydropathic, or other treatment.

But, during scarlatina, changes often arise which urgently demand therapeutic interference, and which are sometimes successfully combated by energetic treatment. Chief among these changes are excessive increase of the bodily temperature and consequent occurrence of symptoms of adynamia and threatening paralysis. Formerly, in such cases, general stimulants were employed, occasionally with the effect of arresting the impending paralysis, as it seemed; but most of the patients died, in spite of the carbonate of ammonia, which was given as a specific in scarlatina maligna. Recently, the knowledge that the hydropaths treat scarlatina very successfully by wrapping the patient in wet sheets, and by cold douches, has greatly increased the popularity of this treatment, which had already been extensively used by *Currie*, and other physicians, in the treatment of asthenic or typhoid cases. And this is the most important therapeutic remedy in malignant scarlatina; and, in cases where there is no dangerous local disease, it often has a surprising effect. The employment of wet sheets and cold douches cannot be replaced by *Schneemann's* treatment of smearing the whole body with grease twice daily during the first three weeks, once daily during the fourth week, although this operation renders the patient more comfortable. I shall not attempt to say whether placing the patient in an empty tub and pouring cold water over him, or wrapping the naked body in wet sheets, deserves the preference; but we should abstract heat by one method or the other every time the temperature of the body becomes excessive, and the symptoms of adynamia reappear. The wet sheets should be reapplied from three to six times, at intervals of ten or fifteen minutes; the patient should then be placed in bed, and left quiet till it is necessary to repeat the process. As even the laity are tolerably familiar with the success of the hydropaths, we rarely meet any great objection to the above treatment, even in private practice. If we should find this objection, large doses of quinine should be given, although I cannot speak of its benefits from personal experience, as I can of those from the energetic abstraction of heat. In desperate cases, where the

above plan fails, we may try to prevent the impending paralysis by powerful stimulants, such as carbonate of ammonia, camphor, musk, etc. The development of angina maligna is not arrested by abstraction of blood, or the local application of cold; but pieces of ice taken into the mouth, and allowed to melt there, have a favorable palliative effect in this as in other severe forms of scarlatinous pharyngitis. As soon as the sloughs have been detached, and foul ulcers are left in the throat, we should proceed to energetic local treatment. We may touch the ulcers daily with a solution of nitrate of silver (℥ j to ℥ ij water), applied by means of a probang, and for the coryza, which almost always coexists, we may inject a weak solution of nitrate of silver (gr. v—x to ℥ ij water) into the nostrils. I have not only seen unmistakable benefit from this treatment in some cases, but I have found that older children submit to the annoying operation without much opposition, because they find that it relieves them. Croup complicating angina maligna indicates the administration of an emetic, and the application of a solution of nitrate of silver to the glottis. We cannot do much against inflammatory infiltration of the lymphatic glands and connective tissue of the neck. According to my experience, abstraction of blood, cold, irritant, and resorbent remedies, cataplasms, etc., have no effect on these hard, indolent tumors, which are covered by normal skin. When they show a tendency to suppurate, and the skin over them grows red, we may apply cataplasms. As soon as there is fluctuation, the pus should be evacuated, to prevent extensive mortification of the tissues, which the pressure of the pus threatens to induce. We sufficiently explained the treatment of croupous nephritis when treating of renal affections, and may also refer to what has been already said for the treatment of scarlatinous dropsy, accompanied with albuminuria. According to my experience, a mild diaphoretic treatment suffices for simple cases of dropsy without albuminuria.

CHAPTER III.

ROSE-RASH—ROSEOLA FEBRILIS.

Cannstatt aptly defines rose-rash as an eruption with red spots, concerning which, from its general symptoms and the affections of the mucous membranes, we are in doubt whether it should be classed with scarlatina, measles, urticaria, or erythema, as it resembles one or other of these diseases in some points, while differing in others. Epidemic roseola arising from infection, which alone we shall discuss here, from unknown causes consists in modifications of scarlet fever or measles. By rubeola scarlatinosa, we mean a scarlet fever where the exanthema

resembles measles, while the high fever, the throat affection, and the dropsy, which often follows, resemble the course of scarlatina. By rubeola morbillosa is meant a form of measles where the exanthema is confluent, and resembles that of scarlatina, while the affection of the respiratory mucous membrane and the escape of the pharyngeal mucous membrane leave no doubt of the morbillous nature of the disease.

CHAPTER IV.

SMALL-POX—VARIOLA—PETITE VÉROLE.

Etiology.—Small-pox spreads solely by contagion; at least, any other cause of its extension and a spontaneous development of variola poison is very improbable, as we can always trace the contagion where there is no peculiar obstacle in the way of our seeking it. Small-pox poison, which we only know from its effects, is contained in the contents of the pustules, and in the perspiration of patients having the disease. This is proved on the one hand by successful vaccination with the contents of the pustules, and on the other by the contagion, which, in most cases, results without immediate contact with small-pox patients. The poison is most active at the period when the clear contents of the pocks begin to turn cloudy. The negative results of attempts at vaccination with the blood and secretions of small-pox patients, seem to prove that the poison is not contained in these fluids. Variola poison is very tenacious of vitality; it is not destroyed by drying; it clings for a long time to objects that have been near a patient with the disease, and, when protected from the air, it remains active for years. The same poison induces the severe forms of variola as well as the mild ones of varioloid. If a healthy person be infected by a patient with varioloid, he may have a severe variola, and the reverse. The different effect of the poison, that is, the unequal intensity of the symptoms, appears chiefly due to the greater or less susceptibility of the patient exposed to the poison. This individual predisposition has always been very unequal; for, in former centuries also, there were some persons who were perfectly insusceptible to variola poison, so that they could expose themselves to it with impunity; there were others in whom the susceptibility was so slight that they were only attacked by the milder forms (varioloid); while, in most persons, it was so decided that, on exposure, they were affected with the severe form of the disease (variola). The liability to small-pox, which, with the above exceptions, is common to all mankind, well and sick, young and old (even the fœtus), men and women, expires al-

most entirely for the rest of life, after one attack. The disease artificially induced by vaccinating with the cow-pock has a similar effect on the predisposition. In some persons it seems to remove the tendency to the disease for life, in others only for a number of years; but the latter class are rarely affected with severe forms of small-pox when exposed to infection. Since, in our day, almost every one has been vaccinated during childhood, it may be readily understood why the number of cases of small-pox is far smaller now than before the introduction of vaccination; and since, after vaccination, any returning disposition to small-pox rarely reaches a high grade, we may also understand why, in the epidemics of the present day, the milder forms of the disease (varioloid) preponderate over the severer forms, which were formerly the most common. At certain times, in more or less extensive localities, from some unknown cause, the intensity of small-pox poison, or the susceptibility to it, is greatly increased; epidemics result. These are more apt to occur in summer, but are not limited to any season. They have a variable duration, and the cases are sometimes malignant, at others very mild, although we cannot discover any cause for this difference.

Anatomical Appearances.—The anatomical changes in the skin, after infection with small-pox, are those of a superficial dermatitis, with a tendency to suppuration. In the milder forms of the disease, the suppuration starts from the cells of the rete Malpighii only; in the severer forms, on the other hand, it attacks the tissue of the cutis, destroys it, and leaves a loss of substance, which is replaced by cicatricial tissue. It is only in the latter cases that evident traces of the disease (pock-marks) remain for life. In the somewhat malignant epidemic that raged in Germany last year, these extensive and deep destructions of the skin, such as used to occur frequently, were rarely seen.

Variolous dermatitis commences with circumscribed hyperæmia of the skin, which, according to *Bärensprung*, extends through the cutis into the subcutaneous tissue. Very soon, elongation of the papillæ in the efflorescence, and, more particularly, swelling of the cells of the rete Malpighii, change the hyperæmic spot of skin into a sharply-bounded nodule, flattened on top, which is not as yet excavated, and contains no fluid, but is perfectly solid. If the process advances (which is not always the case), the outer layer of the epidermis is elevated to a vesicle by a fluid exudation. The contents of this vesicle, at first clear, are soon clouded by the formation of pus-cells, which form from the *young* cells of the rete Malpighii, and the vesicles are thus changed to pustules. Besides the fluid exudation, the pus-corpuscles mixed with it, and the swollen cells of the rete Malpighii (which ofter

form a ring-shaped swelling at the edge of the pock), we find a mesh-work in the pock pustule, which results from *old* cells of the rete Malpighii, compressed by the exudation, particularly of the central cells of the epithelium dipping down between the papillæ. This mesh-work traverses the middle of the pock more particularly, and causes the fan-like formation which has been so much discussed. If the variolous inflammation has not extended to the papillæ, they are compressed and flattened by the pressure of the pock; in such cases the pock-pustules dry up early. New epidermis forms under the resulting scabs; when it is fully formed, the scabs fall off. A shallow excavation is occasionally left at the point where they have been, as the flattening of the papilla is not removed. The case is different when the papilla becomes infiltrated with pus, and breaks down. In such cases the contents of the pustules are changed into a more purulent fluid by the greater admixture of pus-cells, and by the detritus resulting from the breaking down of the papillæ. The pustules become more distended, and thus the umbilications (or shallow depressions in the centre) of some pustules are obliterated. Some pocks burst, and allow their contents to trickle out, others dry up; in the latter case, as the middle part dries first, the umbilication is sometimes renewed. The resulting scabs do not fall off for some time, and they leave depressed, radiated cicatrices, on whose base we may see the dilated openings of the hair-follicles.

The variety in extent and form of the pocks has induced the division into different classes. According as the pustules remain separated by intervals of healthy skin, or touch each other, or, lastly, unite together into vesicles, the cases are divided into *variolæ discretæ*, *cohærentes*, and *confluentes*. Where the pocks do not pass beyond the first stage, so that no vesicles form on the sharply-bounded flattened nodules, it is called *variola verrucosa*. *Variola lymphatica* or *serosa* is where the contents of the pock do not become yellow and purulent, but remain a clear or slightly-clouded fluid; *variola cruenta* is where the pocks are colored red or blackish, by the admixture of blood ("black pox"). A form where the contents of the pock are absorbed, and the empty covering alone remains, is called *variola siliquosa seu emphysematica;* where the pock is filled with a discolored, ichorous matter, and accompanied by gangrene of the skin, the disease is termed *variola gangrenosa.*

We do not find pocks on the serous membranes in autopsy of small-pox patients, but we not unfrequently find the remains of inflammation, with hæmorrhagic exudation. *Sick* found fatty, extensive degeneration in the liver, kidneys, heart, and muscles of patients who had died of variola.

Symptoms and Course.—During the period of incubation there are usually no symptoms either in mild or severe cases. After the inoculation of variola, changes occur at the point of inoculation, even on the third day; but the general health of the patient remains undisturbed, and no other symptom shows the infection till about the ninth day; as the pustule completes its development, the period of incubation terminates. Then there is an eruptive fever, followed by an outbreak of pocks on the other parts of the body, and by other signs of general infection. According to the observations of *Bärensprung* and *Ziemssen*, the period of incubation of non-inoculated pocks lasts twelve or thirteen days.

We shall first describe the symptoms and course of the severe forms, true variola, and close with a description of the milder forms, varioloid.

The first stage of variola, the *stadium prodromorum seu invasionis*, commences with a chill, or with several rigors, which soon give place to a feeling of permanent heat. The pulse is full, and frequent, the temperature increased sometimes to 104° or 106°, the face is reddened, the carotids pulsate strongly; the patients have great thirst, loss of appetite, a bruised feeling in the limbs, and a sensation of pressure and fulness in the epigastrium; the tongue is coated, the taste slimy; nausea and vomiting are frequent, sometimes there is severe epistaxis. Sleep is restless, and broken by dreams. Some patients become delirious; in children we not unfrequently see twitchings, and gnashing of the teeth, or a somnolent condition, which is occasionally interrupted by general convulsions. The high fever and severe constitutional disturbance are not accompanied by such characteristic affections of the mucous membranes as would enable us to decide the nature of the existing infectious disease before us, as we can do in the prodromal stage of measles and scarlatina; but the symptoms above mentioned are almost always accompanied by such severe pain in the sacral and lumbar regions, that we may very strongly suspect the existence of small-pox, when this disease is prevalent. It is not known whether the sacral and lumbar pains, so characteristic of the prodromal stage of variola, depend on hyperæmia of the spinal medulla, on pressure on the spinal nerves, where they escape from the vertebral canal, by the distended venous plexus, or on the excessive hyperæmia of the kidney, concerning which *Beer* has made some important suggestions in his valuable examinations into the connective tissue of the kidneys. After imperfect remissions during the second and third day, the fever and accompanying disturbances usually increase, and attain their height on the evening of the third day. Generally, the intensity of the symptoms in the prodromal stage is in direct proportion to the subsequent eruption,

and the highest fever and most severe disturbances usually precede confluent small-pox. But this is not always the case; sometimes a severe prodromal stage is followed by a scanty eruption; in other cases, a very mild prodromal stage precedes a confluent eruption. Deviations in the duration of the prodromal stage are rarely observed, and still more rarely it is entirely absent, and the eruption is the first symptom.

The second stage, *stadium eruptionis*, almost always begins with the appearance of the first papules in the third exacerbation of the fever. If the eruption be very copious, the red points lie close together, and unite at some places, as they do in measles, or by a general confluence they cause a regular redness, and granular swelling of the face; if the eruption be scanty, the different nodules remain isolated, and are separated by more or less extensive patches of normal skin. The next day the eruption usually spreads from the face to the neck, breast, and back, and on the third day to the extremities. The number of papules on the body and extremities is proportionately far less than on the face; so that, even when the disease is confluent on the face, the pustules on the body and extremities usually remain discrete, or at most would be called variolæ cohærentes. The points gradually become large, blunt conical nodules; the second or third day these change to vesicles, and the third or fourth to pustules, the contents becoming cloudy and purulent. The pocks appear late on the extremities; they also develop late; on the face, the papules have become larger, and changed into vesicles, or their contents have become cloudy when the first nodules appear on the extremities. The eruption on the mucous membrane begins at the same time it does on the skin; but we do not generally notice it till later, as it at first causes little trouble. The pocks in the mouth induce increased flow of saliva; those in the pharynx, difficulty of swallowing; those in the air-passages, hoarseness and cough; those on the conjunctiva, lachrymation and photophobia. The fever, the severe pains in the sacral and lumbar regions, and the constitutional disturbance, which attain their height toward the end of the prodromal stage, always remit when the eruption comes out, decrease still more as it extends to the body and limbs, and toward the end of the eruptive stage the patient usually feels quite well. He is then encouraged because he thinks he has passed through the worst. A few years since, a patient with confluent small-pox presented himself at my clinic, who, after lying in bed for three days at Esslingen, with severe fever, had walked the five miles from Esslingen to Tübingen without difficulty. In confluent small-pox the eruptive stage is usually shorter, the exanthema spreads over the body more rapidly, the contents of the pocks become purulent

earlier, and from the confluence of the pustules the face often looks as if it were covered with one large bladder of matter. The affections of the mucous membrane also, which in variola discreta do not usually cause much trouble at this time, are also accompanied by very painful symptoms, severe salivation, dysphagia, croupy cough, great photophobia, and by ischuria and burning pains in the external genitals. The high fever and severe constitutional disturbance of the prodromal stage remit during the eruptive stage of confluent small-pox also, but only to a moderate extent; the fever never disappears entirely, as it does in discrete variola, nor does the patient feel well toward the end of the eruptive stage, as he does in the latter form.

The third stage, *stadium suppurationis seu maturationis*, begins about the sixth day after the first appearance of the eruption, and about the ninth day after the first symptoms of fever. The pocks become larger; the bluntness of their summit gives place to a hemispherical shape. If they be punctured, their thick purulent contents escape all at once, because of the atrophy of the partitions, which previously let only part of the contents escape on puncture. The skin around the pocks swells decidedly, and becomes dark red; even in discrete variola the red areola surrounding one pock unites with that around the next. The redness and swelling thus become diffuse. The patients are greatly disfigured, and complain of severe, tense, pulsating pain in the reddened skin, which is covered with large pustules. More or less of these pustules rupture, and their contents flow over the surface, where they dry into crusts, which are at first yellow, subsequently brown. In these changes, also, the pocks on the body and extremities are one or two days behind those on the face. The pain and distress induced by the intense dermatitis are increased, during the suppurative stage, by the annoying symptoms due to the eruption on the mucous membrane. The saliva trickles constantly from the mouth of the patient, swallowing becomes almost impossible, the nose is stopped, the voice inaudible, the cough distressing and hoarse, the eyes, dark red and filled with muco-pus, burn and are very sensitive to the weakest light. The eruption in the vulva, vagina, and urethra, comes later than that in the mouth, pharynx, and larynx; hence the tense, burning pain in the external sexual organs and the ischuria are most severe after the salivation, dysphagia, and laryngeal symptoms have subsided. The fever, which had moderated or even disappeared during the eruptive stage, exacerbates during the suppurative stage, or begins anew with repeated chills. This fever is generally called the secondary or suppurative fever, and it really seems as if the secondary fever did not directly depend on the variolous infection, but were chiefly or solely due to the dermatitis. The more severe and malignant the inflamma

tion of the skin, the more intense is the fever; the dermatitis and the fever attain their height together, and the decrease and disappearance of the eruption go hand in hand with the decrease and subsidence of the suppurative fever. In many cases the suppurative fever becomes dangerous, because the bodily temperature rises so high as to induce the symptoms of adynamia and fatal paralysis (asthenic, nervous, typhous small-pox). In such cases, just as in asthenic measles and scarlatina, the symptoms of adynamia are not unfrequently accompanied by those of an acute hæmorrhagic diathesis; the contents of the pock become bloody, and petechiæ appear between the pocks. Occasionally, also, there is profuse epistaxis, more rarely hæmatemesis, bronchorrhagia, or metrorrhagia (hæmorrhagic or septic small-pox). In some rare cases, during the excessive adynamia of the suppurative stage, the inflammation of the skin increases to partial mortification of the inflamed tissue; gangrene of the skin results, and the pocks fill with a discolored ichor. Such patients almost always die early with the symptoms of excessive prostration (gangrenous small-pox). Besides the adynamia induced by the high fever, and besides the occurrence of a hæmorrhagic diathesis or of cutaneous gangrene, which favor the threatening paralysis, the suppurative stage is especially dangerous on account of the localization of the variolous process on the serous membranes and in the parenchymatous organs, which occurs most frequently during this stage, and from the frequent increase of the affection of the mucous membrane to croupous and diphtheritic inflammations. Thus, in many cases, increased dyspnœa, sharp pain in the side, tormenting cough, bloody expectoration, as well as the physical signs of condensation of the lung, show the occurrence of pneumonia. In other cases the subjective and objective signs of pleurisy accompany the above symptoms of the stage of maturation. The symptoms of the disease are also variously modified by the occurrence of suppuration of the joints, suppurative periostitis, subcutaneous, and intermuscular abscesses, inflammation and suppuration of the lymphatic glands, panophthalmitis which begins as a hypopion and may be readily overlooked, as well as by the symptoms of pericarditis, meningitis, or of pyæmia from absorption of ichor. It is chiefly in confluent small-pox that the fever assumes an asthenic character during the suppurative stage, and that unusually dangerous local diseases of the internal organs occur. Moreover, in confluent small-pox, during the stage of maturation, the eruption on the mucous membrane of the pharynx and larynx readily becomes complicated with croupous exudation and œdema of the glottis. Hence, this form of the disease is correctly considered very malignant; and confluent small-pox is often regarded as identical with malignant.

The fourth stage, the *stadium exsiccationis*, usually begins on the eleventh or twelfth day. Part of the pustules, which have remained unruptured up to that time, burst, and their contents, which are thrown out, dry to soft, yellow crusts, which subsequently become hard and brownish. The other pocks lose their tension, become brown in the middle, dry, and sink in; gradually the contents and wall of the pock dry into a round scab. In proportion as the formation of the crusts progresses, the parts around lose their redness and swelling; the patients again become recognizable; the tense, pulsating pain subsides, and gives place to a very annoying itching, so that the patient finds it very difficult to avoid scratching. In the stadium exsiccationis it is usually most evident that the pocks on the extremities are of more recent origin than those on the face; for just at the time the painful tension and the disfigurement of the face are disappearing or are already gone, the patients complain of unbearable pain in the swollen fingers and toes. The scabs are detached very irregularly; at the points where there has been no ulceration of the skin (and these are more or less frequent, even in the most malignant cases) the crusts do not generally remain attached more than three or four days; on the other hand, at points where the ulceration has entered deeply into the cutis, the crusts remain attached longer; as the suppuration continues, they become thicker and horny. The crusts that fall off early leave red spots, which are somewhat elevated as long as the skin remains swollen, and which, after a time, disappear without leaving a trace, or else change to slight depressions. On the other hand, when the crusts that have remained adherent for a long time at last fall off, round deep cicatrices are left, these have inverted edges and a punctate, seamed base. They are at first red, then become very white, and last during life. The symptoms induced by the eruption on the mucous membrane disappear when the redness and swelling of the skin subside. The salivation, dysphagia, hoarseness, cough, photophobia, and ischuria, all cease gradually. At the commencement of the stadium exsiccationis the fever continues, but is moderated (exsiccation fever); but the more the formation of the crusts progresses the more the fever decreases; the pulse becomes quiet, the skin is covered with perspiration, the urine deposits a sediment, the general health becomes normal. In confluent small-pox, which usually extends more in depth, as well as superficially, than other varieties, during the drying stage thick, dark crusts form, which cover the face like a mask. The irregular crusts constantly increase on account of the continued suppuration, and cracks form in them, from which the pus trickles out. Finally, the crusts fall off in large pieces, and extensive irregular losses of substance are left, instead of small round cicatrices, as after variola dis-

creta. In such cases, just as after deep burns, the retractions of the cicatricial tissue readily induce tense cicatrices, or bridge-like strings, ectropion, distortion of the mouth, etc., which cause the most hideous deformities. Not unfrequently, a previously beautiful countenance is rendered disgustingly ugly by confluent small-pox. Salivation, hoarseness, cough, and the other symptoms of variolous affections of the mucous membranes, disappear far more slowly after confluent small-pox than after the discrete form. And the affections of important organs that have been caused by the infection during the eruptive stage have not usually terminated in the suppurative stage. At this time the patient often has subcutaneous and intermuscular abscesses, boils, and other localizations of the variolous process, but especially pyæmia. Hence, too, the secondary fever is usually more severe and continued in confluent small-pox than in the discrete, and even in the most favorable cases a long convalescence follows the severe disease.

We may describe *varioloid* very briefly, because its symptoms differ from those of variola only in degree. The prodromal stage is the same as that of small-pox, except that it is shorter and less intense. But there are numerous exceptions to this, a severe and protracted prodromal stage being sometimes followed by varioloid instead of by variola. The appearance of an erythematous rash, forming large red spots, or of a diffuse scarlatinous redness over a large part of the body, particularly the lower part, is somewhat characteristic of varioloid, because it occurs more rarely in variola. This exanthema precedes the varioloid eruption from twelve to twenty-four hours. In the eruptive stage, which usually begins with the third exacerbation of the fever, the eruption does not spread from the face to the body and extremities quite so regularly as it does in variola. The eruption usually ends in twenty-four or thirty-six hours. The pocks are generally less numerous than in variola; they rarely become confluent, and then only at certain spots. The papules become vesicles, and the vesicles pustules, much sooner than in variola; the form of the efflorescence offers no peculiarities. In varioloid also, the mucous membranes are affected, and we have salivation, difficulty of swallowing, hoarseness, cough, etc. As the eruption terminates, the fever usually disappears entirely, and the patient almost always feels quite well, except suffering some inconvenience from the affection of the mucous membrane. The most important differences between varioloid and variola occur in the stage of maturation; for it is then most evident that in the milder form the dermatitis is more superficial, while in variola it affects the parenchyma of the cutis deeply. During the suppurative stage the pocks are filled with thick fluid pus, they become larger, and hemispherical; some of them burst, and let out their contents; but the areola around them is

smaller, and less swollen, consequently the tension of the skin, the pain, and deformity, are always less than in variola. As the dermatitis is not so severe, the secondary fever is less, and, when the eruption is very scanty, the stage of maturation may run its course almost without fever. During the suppurative stage variola and varioloid differ less in regard to the affections of the mucous membranes; for the photophobia, dysphagia, cough, and hoarseness in varioloid often trouble the patient as much as they do in variola; on the other hand, croupous and diphtheritic inflammations of the mucous membranes, and malignant affections of the internal organs, are very rare. The suppurative stage usually passes into the stage of desiccation by the fifth or sixth day of the eruption. But few pustules rupture; most of them dry up with their contents; a brown, dry spot forms in the centre, gradually becomes larger, and as it reaches the periphery, the pock changes into a brown scab; most of the scabs fall off in three or four days, and leave red, somewhat prominent spots. The contents of some of the pocks are not thickened, but are reabsorbed, and, instead of a slough, the wrinkled, dry envelope is thrown off; this course is seen quite often, especially in the pocks on the extremities. But, as, in the severest cases of variola, some pocks always spare the tissue of the cutis, and do not leave a scar, so, in the mildest cases of varioloid, single pocks, which exactly resemble those of variola, often cause a loss of substance, and leave cicatrices. If we group the peculiarities that distinguish varioloid from variola, we should mention especially: 1. The short duration of the different stages, and of the entire disease. 2. The mildness or total absence of the secondary fever. 3. The escape of the cutaneous tissue, and the recovery without cicatrices. 4. The slighter mortality. In the times when, during small-pox epidemics, variola prevailed, or occurred exclusively, a third, or in some epidemics even a half, of the patients died. Of late, as the disease almost exclusively affects persons who have been vaccinated, and consequently varioloid is in excess, the mortality is very low; scarcely four or five per cent. of the patients die.

Treatment.—Prophylaxis demands vaccination or revaccination. All objections to vaccination, even if well founded, would have to give way to the facts, proved by statistics, that in the last century one-tenth of the population died of small-pox (about 400,000 people dying of the disease every year in Europe), another tenth were disfigured by the disease; and that, since the introduction of vaccination, the general mortality is less, and that of small-pox is reduced to a minimum. During an epidemic, persons who have been vaccinated, but in whom the vaccination has not yet taken, are attacked by small-pox, and the two diseases run their course at the same time without modifying each

other. These cases show that vaccination during the period of incubation of small-pox cannot cut short the disease, and also that the susceptibility to vaccination is not lost during the period of incubation. To avoid vaccinating persons who are already in the incubation stage of variola, it is well for a physician to vaccinate without delay all of his patients, where he considers the operation necessary, as soon as he knows of the appearance of small-pox in the vicinity. Inoculated small-pox generally runs a milder course than that due to infection, it is true; but the exceptional cases where the inoculated variola runs a malignant course, and ends fatally, are still so numerous that at present we should not think of inoculation, as we have vaccination to rely on. Besides vaccination and revaccination, we should not be deterred by any personal considerations from exercising the most stringent rules for the isolation of small-pox patients, even if they should be very annoying for the patient and his friends.

The treatment of small-pox can only be symptomatic, as we know of no remedy for cutting short the disease, and averting dangerous accidents. In the prodromal stage the patient should be kept moderately cool, the temperature of his room should be about 60°, the bedcovers should not be too heavy; we should not dose him with warm tea, but let him have cold water or lemonade, absolute diet, and, where there is constipation, we may order enemata of three parts of water to one of vinegar. If the prejudices of the patient render it necessary to give some medicine, we may order a solution of nitre. Even in severe congestion of the head, with a full pulse, we should avoid bleeding, which was formerly so popular in inflammatory small-pox, and limit ourselves to the application of cold to the head, and repeated ablutions of the body with cold water. The administration of camphor and other remedies, for causing derivation to the skin and hastening the eruption, formerly so much in vogue, is very objectionable. During the eruptive stage we should especially attempt to prevent the development of a severe eruption on the conjunctiva. This may be done by the assiduous employment of cold water compresses, or, still better, by compresses moistened with a weak solution of corrosive sublimate (gr. j to water ℥ vj). Cold and the mercurials are also much used for the prevention of deep destruction of the skin, and disfiguring cicatrices. For this purpose it is best to cover the face (which is the only part usually protected) with a mercurial plaster, emplastrum de vigo, and to leave it on for several days. *Skoda* prefers compresses moistened with solution of corrosive sublimate (gr. ij—iv to water ℥ vj) to mercurial plaster, which induces an injurious elevation of temperature. *Hebra* rejects both mercurial plaster and solution of corrosive sublimate, as well as collodion, and touching the individual pocks with nitrate of

silver, which have also been recommended. He has come to this decision from the observation in his wards that the pocks do not leave cicatrices any oftener since he has stopped employing these remedies, than when he used them. *Hebra* applies only cold-water compresses; while the skin is tense, these relieve the patient, although they cannot protect the skin from destruction. During the eruptive stage, the chief attention should be directed to the fever, for the patient may be much endangered if it become severe. Unless the temperature rise very high, we may abstain from treatment; if it do rise high, we should give large doses of quinine. In the stage of desiccation, we should let the patient have an easily-digestible but nutritious diet, and a little wine; for the exhausted strength requires building up, and the frequent pulse subsides more readily under the careful supply than under the continued withholding of the nutritive material necessary for the restitution of the body. The patient should be warned against scratching off the crusts, and we should take measures to prevent his doing so involuntarily, while asleep. If the crusts be firmly adherent, and especially if suppuration under them continue, we should employ cataplasms. Among the other symptoms requiring especial attention during variola, are the troubles from the affections of the mucous membranes. We cannot hope for an abortive treatment of the pocks on the mucous membrane, except in those in the mouth; and, even there, astringent mouth-washes, and touching the individual efflorescences, are of no material service. If dangerous dyspnœa, and other symptoms of croupous laryngitis occur, we may give an emetic of sulphate of copper, and attempt to apply a solution of nitrate of silver through the entrance of the glottis, by means of a probang. In œdema glottidis, if we cannot relieve the patient by scarification, we should perform laryngotomy. Inflammation of the serous membranes, and of the parenchymatous organs during small-pox, may require venesection, especially when, during pneumonia or pleurisy, collateral œdema induces difficulty of respiration; but it should only be employed in cases of absolute necessity, for experience shows that abstraction of blood is badly borne in all infectious diseases. In other respects pneumonia, pleurisy, and other local affections induced by variola, are to be treated according to the rules given when speaking of the primary forms of these diseases. Abscesses of the cellular tissue and suppurating lymphatic glands should be opened early.[2]

CHAPTER V.

COW-POCK—VACCINA.

Etiology.—Vaccina is an excessively infectious disease. The remarkable fact, that, even at the present day, extensive epidemics of small-pox occur in spite of most persons being vaccinated, or even in many cases revaccinated once or oftener, has induced me to make some very careful observations regarding the duration of the protective power of vaccination. I have not yet completed this work, but have gone far enough to satisfy myself that the protection against variola afforded by vaccination is for a far shorter period than is generally believed.

On the presumption (which is certainly correct) that a person who takes vaccina after vaccination would have taken variola or varioloid if he had been exposed to infection, I have made a large number of observations on revaccination. Through the politeness of Surgeon-General *Von Klein* and Dr. *Von Köllreutter*, in Stuttgart, among other cases, I have had the opportunity of following, day by day, the effect of revaccination in five hundred recruits. In these cases, where I had perfect control, I satisfied myself that the number of the recruits in whom normal cow-pocks developed was proportionally small, and corresponded very nearly with the number given by other observers. But it also showed that the number where there was no result was also slight. In most cases, a few days after the vaccination, there were redness and infiltration of the skin, papules, vesicles, etc. Among the latter class, in many cases, eight days after the operation, the inflammation has run its course, and, if the case has not been watched during the interval, it would be classed among those where the vaccination had no effect. These inflammations at the point of vaccination may have been the result of the wound from the lancet, and of the foreign body introduced under the epidermis through it, or a result of scratching; but there was also a possibility that it was due to incomplete vaccina running a rapid course, and holding the same relation to ordinary vaccina that varioloid does to variola, and which consequently has been called vaccinoid or varioloid-vaccina, in opposition to variola-vaccina. To settle this question, I first vaccinated a number of persons with the contents of blisters and of pustular eruptions. In none of these persons was the like result produced. I also revaccinated a number of my pupils and house-patients, in whom, at the first attempt, this modified result had occurred, and in them also the effect failed. These facts make it appear to me very probable that, at the time of a small-pox epidemic, in spite of the common vaccination and revaccination, the

number of persons liable to variola or varioloid is quite large; and, although I have arrived at no conclusion as to the duration of the protective power of vaccina, I think I may still advise physicians to revaccinate all their patients at the outbreak of an epidemic, no matter what the length of time since their last vaccination. The contents of the vaccina pustule are the only known vehicle of the contagion; the emanations from the skin and lungs, which convey the variola poison, do not carry vaccina poison; at least, a well person is never infected by entering the atmosphere of a person or beast with vaccina. A second important difference between variola and vaccina poison is, that the latter only induces an eruption at the point where it was introduced, never at remote parts, as the former does. It is not necessary to decide the question whether this difference depends on the fact that vaccina poison is of a different nature from variola poison, or whether the latter is weakened by being transferred to another species of animal, being more imperfect and less active when reproduced in the cow. The predisposition to vaccina is very general, and it is rare for a person vaccinated for the first time to escape the disease. A single attack sometimes removes the liability to the disease for the rest of life; but, in the majority of cases, this immunity only lasts a number of years, and then ceases. The important discovery, that having had vaccina removes the susceptibility to small-pox just as it does to a second attack of vaccina, is still more important, since it has been shown that even this protection is only temporary, and that vaccination must be repeated from time to time, if we would prevent a return of the liability to small-pox. Leaving out of consideration certain foolish objections, that have been advanced against vaccination, it cannot be denied that it sometimes endangers life, and in other cases leaves permanent impairment of health, especially cutaneous eruptions, and other scrofulous affections. The hypothesis that scrofula was transferred, by the vaccination, from one child to the other, is false, as may be proved. Sometimes children become scrofulous after vaccination, although the lymph have been taken from the arm of a perfectly healthy child; and sometimes children remain perfectly healthy after being vaccinated with lymph from a decidedly scrofulous child. The occurrence of scrofula after vaccination seems to be due to the debilitating influence of the fever accompanying the vaccina, and the prominence of the exanthema among these scrofulous affections appears to depend on the disease of the skin, artificially induced at the point of vaccination. At least other febrile diseases, as well as all debilitating affections, occurring in young children with a tendency to scrofula, have the same influence in developing this disease that vaccina has. And we know that blisters, and other irritants to the

skin, not only induce inflammation at the point of application, but also increase the predisposition to other cutaneous affections. Many children, who have never previously suffered from any exanthema, are affected for months with moist eczema of the face, after having their ears pierced, as well as after vaccination. But it is only in rare cases that the occurrence of scrofulous affections is due solely to vaccination, and is not influenced by other causes, such as weaning and teething, which usually take place about the time that vaccination is done, and it is still rarer for life to be endangered by the operation. Complete ignorance of statistics of mortality, which show a decided decrease of mortality since the introduction of vaccination, must be the only excuse for urging these exceptional cases as grounds against vaccination. It may be readily seen why more children die of measles, scarlatina, croup, and hydrocephalus, since small-pox leaves a larger number for these diseases to attack, as it were. But the slight increase of mortality in the above diseases does not, by any means, equal the diminution of mortality in variola. Unless the occurrence of a small-pox epidemic throws all other considerations into the shade, I do not vaccinate weakly children, inclined to scrofula, during their first year, but wait till the second or third, after the teeth have developed, because I am satisfied that, in such children, very much depends on protecting their first development from injurious influences.

Anatomical Appearances.—The anatomical changes in the skin after the first vaccination exactly resemble those in variola, except that they are confined to one point. The third day after the vaccination there is a small red nodule at the point of operation; the fifth or sixth day this changes to a vesicle; by the eighth day the vesicle attains the size of a lentil, and has a central depression, the umbilication, and a cellular formation. The ninth day the contents of the pock become cloudy and whey-like, while a broad, dark-red halo develops around their periphery; on the tenth day they become purulent; from this time the pock begins to dry up, rarely rupturing. Toward the end of the third week, or even later, the crust falls off, and leaves a somewhat excavated, round, white cicatrix, whose base is stippled and ridgy. The umbilication of the vesicle, according to *Simon*, depends on the puncture made during the vaccination; as a result of the inflammation caused by the puncture, there is an adhesion of the epidermis to the cutis, hence, when a serous liquid is subsequently effused between the two membranes, the epidermis does not become loosened at the point where it is most firmly attached; this hypothesis is supported, among other points, by the fact that the form of the umbilication corresponds to that of the wound: if we have vaccinated through a puncture, the depression is small and round; if

through an incision, there is an elongated depression along the middle of the oval pock. The changes that occur at the point of vaccination, in persons whose susceptibility to vaccina is diminished, are very varied. They always take place sooner, and run their course more rapidly, than in normal vaccina. In some cases we see nodules, or wart-like swellings, surrounded by a dark-red and infiltrated areola, which either do not develop further, or else change to vesicles, whose contents quickly dry up. In other cases, on the reddened and infiltrated spots we see no efflorescence; in still other cases, there is a furuncular inflammation of the skin, resulting in superficial ulceration. To determine, in any case, whether this inflammation be modified vaccina, we would have to vaccinate a child, who has not yet been vaccinated, with the products.

Symptoms and Course.—Regarding the symptoms and course of normal vaccina, a short description may suffice. There is no premonitory stage. In the eruptive stage, the anatomical changes in the skin, above described, are the only symptoms of the disease. In the stage of maturation the inflammation causes severe tense pains, and hinders motion of the affected arm. The lymphatic glands in the axilla often swell. Sometimes an eruption, resembling lichen, or eczema, instead of the pock eruption, appears in the vicinity of the vaccina pustule and at other parts of the body. While fever never occurs at the commencement of vaccina, the stage of maturation is always accompanied by a secondary fever. In incomplete, modified vaccina, the inflammation of the skin sometimes causes insupportable itching, sometimes tense or burning pain. From numerous measurements of temperature, I have satisfied myself that the patients have a fever which is not at all in proportion to the intensity and extent of the dermatitis. Where the local symptoms were very slight, I have noticed a temperature of 104°. But, from the less extent of the dermatitis, the fever never becomes so high as in variola; it can only prove dangerous to very weakly children, and even then only in rare cases. It is rare also for dangerous erysipelatous or pseudo-erysipelatous inflammation of the arm to occur during the stage of maturation.

Treatment.—In the following paragraphs we have to treat not so much of a cure of vaccina, as of conducting the disease to a proper termination. The most important rules to follow, in vaccinating, are: 1. To take the lymph from a healthy child, who has not previously been vaccinated. Little as we believe in the transfer of dyscrasias by vaccination, we still consider it foolhardy and improper to make useless experiments as to whether this view is correct. Experience shows that the lymph taken from the vesicles of revaccinated persons is much less certain to produce normal pocks than that taken from per-

sons vaccinated for the first time. 2. We should take this lymph, the seventh or eighth day after vaccinating, from pocks which are perfectly normal as regards size, form, and areola. The lymph from older or younger pocks, or from those in any way abnormal, is less certain. 3. When possible, we should vaccinate from arm to arm. If obliged to use old lymph, that which has been preserved in glass tubes, with the ends sealed, is the best. 4. We should vaccinate through punctures made with the lancet or vaccinating needle, and should choose a part of the arm which would be covered even when short sleeves are worn. About five punctures should be made in each arm; sufficient space should be left between the punctures to prevent the confluence of the pocks or their areolæ. 5. Except during the prevalence of a small-pox epidemic, we should only vaccinate healthy persons. If the vaccination do not succeed, we should repeat it after a few months. 6. When an epidemic of the disease breaks out, it is well to revaccinate persons who have not been vaccinated for seven years or more. 7. We should protect the pocks from pressure and friction, and should especially guard against children scratching their arms; and we should keep the patient in his room during the maturating fever. If there be much inflammation around the point, we may apply compresses wet with lead-water; if ulcers remain, use cataplasms.

CHAPTER VI.

WIND-POX, WATER-POX, SHEEP-POX, VARICELLA, CHICKEN-POX.

Etiology.—Many dermatologists consider varicella as identical with variola and varioloid, and as being the mildest form of small-pox. From this view, the almost exclusive occurrence of this disease in children is explained by saying that in them, from the short time which has elapsed since vaccination, although the liability to small-pox is not entirely gone, it is reduced to a minimum; while in older persons the liability to small-pox is usually so much reëstablished by the long period that has elapsed since vaccination, that, when exposed to the contagion, they are affected with varioloid. The falseness of this view may be shown by striking examples. In the first place, the frequent occurrence of varicella in children that have never been vaccinated, speaks against the identity of varicella and variola poison. Why should persons who might be supposed to have the greatest predisposition to small-pox be so often attacked by the mildest form of the disease? But experience also shows that varicella offers no immunity to vaccina or variola; on the contrary, many children who have just recovered from varicella are successfully vaccinated, or, during an epi

demic of small-pox, may take variola or varioloid; this is in direct opposition to the almost absolute protection afforded by an attack of either of the latter diseases to another attack. Lastly, cases also occur where children who have been vaccinated or had variola a few weeks previously, are attacked by varicella. The above facts render it certain that varicella is not the mildest form of small-pox, but is a disease *sui generis*. The cases where persons with varicella are said to have infected others with variola or varioloid, and vice versa, appear due to mistaking mild cases of varioloid for varicella. The infectious origin of varicella may almost always be traced; but all those infected, whether vaccinated or not, are attacked by varicella, not by variola or varioloid. Varicella usually occurs in more or less extensive epidemics, which occasionally accompany, precede, or follow small-pox epidemics, but in other instances coexist with epidemics of measles or scarlatina; varicella not unfrequently occurs sporadically also.

Anatomical Appearances.—The exanthema begins as small, red, distinct spots, which, after a few hours, are changed to limpid vesicles of the size of a pea or lentil, by a copious serous effusion between the cutis and epidermis. These vesicles have neither a central depression nor a cellular formation. After a time their contents become whey-like, but never purulent. Thin crusts form from the dried vesicles, which fall off after a few days, without leaving a cicatrix. From the form of the vesicles, the different varieties, varicellæ globulosæ, ovales, lenticulares, coniformes, and acuminatæ, are distinguished. We often see, especially when the eruption is very extensive, that, besides numerous varicella vesicles running the usual course, a few filled with pus (varicellæ pustulosæ) acquire the appearance of variola pustules, and even leave cicatrices. Since the form of variola pustules is not specific, but exactly resembles that of some erythema pustules, we should not attach too much importance to the external resemblance of some of the efflorescences in varicella to those in variola, and consequently consider the two diseases as identical.

Symptoms and Course.—In some cases the appearance of the eruption is the first symptom of the disease. Even the most careful and anxious mothers often notice no prodromal stage, and assure us that, the day before the eruption appeared, the child was perfectly well. More rarely the exanthema is preceded for a day or two by slight fever and general discomfort, derangement, loss of appetite, headache, etc. The varicella spreads over the body without any regularity. The eruption is usually thickest on the back and breast. The face occasionally remains perfectly free. Individual vesicles develop in from six to twelve hours; their contents become cloudy the second day, and dry up the fourth. It is rare to have only a single crop of

vesicles; new crops usually appear for several days, so that the disease is often protracted for a fortnight, or longer; and in the later stages we find dried vesicles and fresh ones alongside of each other. Rarely vesicles form on the mucous membrane of the mouth and fauces, and they soon change to small, round ulcers. There is no constitutional disturbance.

Treatment.—Treatment is scarcely necessary in varicella, as the disease is not dangerous, or very inconvenient, and always terminates in recovery, after lasting a week or two. Nevertheless, it is advisable to protect the patients from all injurious influences while the affection lasts, to keep them in their chamber, and to regulate the diet.

CHAPTER VII.

TYPHUS FEVER—EXANTHEMATIC TYPHUS—PETECHIAL TYPHUS, SPOTTED FEVER.

Etiology.—Exanthematic typhus (morbus acutus, febrilis, contagiosus, exanthemate proprio ac eminenti systematis nervosi passione stipatus, *Hildebrand*) is very closely related to the previously described infectious diseases, measles, scarlatina, and small-pox, by its contagiousness, and by the local affection of the skin in the shape of an extensive exanthema; while, on the other hand, its symptoms correspond so closely with those of abdominal typhus, that many authors consider it a variety of the latter. The contagiousness of exanthematic typhus can only be doubted by those who have had the opportunity of observing the disease in large epidemics solely. When an epidemic disease first affects a large city, or still larger district, it is almost impossible to decide whether the disease be transferred from one person to another, or if different individuals be affected independently from having been exposed to the same causes of injury. The case is quite different when the disease attacks smaller places, where it may readily be watched and its extension observed. In such cases there is a more favorable opportunity of observing the contagious or non-contagious nature of the malady. All observers who have had such an opportunity in exanthematic typhus agree that the power of contagion is just as evident, in this disease, as in any other. Among the numerous observations tending to prove the contagiousness of exanthematic typhus, I may mention two of my own, which are particularly striking. In the year 1854 two typhus patients were received into the Magdeburg hospital from the prison, which was much crowded. For months previous there had been only a few cases of abdominal typhus, and not a single case of exanthematic typhus in my wards. Eight

days after the reception of these patients, two patients that had lain alongside of them were attacked by the same disease (one of them had been received into the hospital for intermittent fever, the other for epilepsy); in a short time the attendants who had taken care of the patients were similarly attacked. After the isolation of these patients, all the other persons in the hospital escaped the disease; nor were there any other cases in the city at this time. In March, 1855, a tradesman from Heiligenstadt was attacked with exanthematic typhus while away from home; he was received into my ward; a year had almost elapsed since the appearance of the last case there. Eight days after the reception of this patient, a blacksmith's apprentice and a mechanic, lying next to him, were attacked by the disease. After these patients were isolated, my assistant, a washer-woman, and every one who had been chosen to attend the patients, were also attacked. It was not till a convalescent from exanthematic typhus was placed as the sole attendant for the typhus patients, that new cases ceased to appear. (These patients were nursed with great conscientiousness and self-sacrifice by the convalescent, who, for twenty years previously, had been in chains for the murder of a clergyman, and had only obtained his liberty a short time before.) The contagion is contained in the atmosphere about the patient, in his clothes, bed linen, and other property. Hence the treatment and nursing of patients with exanthematic typhus is much more dangerous than is the case with patients having cholera or abdominal typhus. Like measles, the disease may be carried by persons who do not themselves become affected. The more patients are crowded together, the more intense the contagion becomes; this fact agrees perfectly with the recently-adopted view of a contagium vivum (see page 673). A place of a certain size, where many patients produce the germs of the disease, is more thoroughly filled with the poison than one where the germs are only produced in the body of a single patient.

I do not agree with most recent authors in considering the question settled in regard to whether exanthematic typhus be exclusively propagated by contagion, or if it be also due to *miasm*. There is no solid ground for such assertions as this, "that a disease is either *only* miasmatic, or *only* contagious," and even the persons making them do not always strictly adhere to them. When one acknowledges that the germs of cholera probably develop from diseased rice, and are thence brought to us by cholera patients and their dejections, he cannot deny the possibility, and even probability, that the germs of contagious diseases native among us may, under favorable circumstances, develop and increase outside of the human body. The hypothesis of a "spontaneous generation" of infectious diseases, in the sense that their cause is a new agent induced by injurious influences, is, of course, to

be rejected, for, in that case, we should be inferring a *generatio aequivoca*, which has been disproved; but it has not by any means been proved that, besides purely miasmatic affections, whose germs develop outside of the organism and are not reproduced in the bodies of patients suffering from them, there are no *miasmatic-contagious* diseases, whose germs develop outside of the body, but are also reproduced in the bodies or dejections of patients suffering from them. I know of no absolute objections to this theory, and a series of facts agrees better with it than with the unproved assertion that there is no miasmatic-contagious disease. We not unfrequently see the disease in question appear under circumstances which render origin from contagion very doubtful; for instance, in ships at sea (ship-fever), in carefully-guarded prisons (jail-fever). On the other hand, under certain circumstances, which appear favorable to low organisms and to their reception into the body, as in years of famine, where bad, spoiled food is eaten (famine typhus) and in over-filled lazarettos, where the air is loaded with exhalations from the patients (lazaret-fever), exanthematic typhus occurs so frequently, that, where these circumstances prevail, its appearance may almost be predicted. Of course, this does not prove that the body of a patient diseased by the introduction of these germs may not be a more favorable soil for its development and reproduction than decomposing flesh, or the air of a lazaretto overloaded with excretions, and that the disease may not only continue to exist, but to extend and even spread greatly, long after the disappearance of the circumstances which favored the development of the germs of the disease outside of the body. If the above-mentioned assertion, that the cholera-germ is developed on dried rice, should be confirmed, it would be still more probable that the germ of exanthematic typhus developed outside of the human body in decomposing animal matter, and when circumstances were peculiarly favorable, increased so as to become dangerous to man. The susceptibility to the miasm or contagion of exanthematic typhus is very general. Males and females, strong, healthy persons and weak, sickly ones, are about equally liable to the disease. Only early childhood and extreme age are usually exempt. Excessive exertion and other debilitating influences appear to increase the predisposition. One attack seems to destroy the susceptibility to the affection.

Griesinger and *Hirsch* speak of the geographical extension of exanthematic typhus in Europe thus: from the beginning of the sixteenth to the end of the eighteenth century, this disease extended over all Europe as the common form of typhus; during the war, at the commencement of this century, it attained its height. After that time it was so rare on the continent, that there was a current belief that

typhus never occurred without ulcers in the intestines. It is only in the last ten years that the epidemics in Upper Silesia, the fatal epidemics during the Crimean War, and recently the epidemics in East Prussia, etc., have dispelled this error. In the British Islands, and in certain places in middle Europe, exanthematic typhus is the endemic form of the disease. In Southern Europe, Lower Italy, the Oriental countries, Hungary, etc., exanthematic typhus occurs, sometimes alone and sometimes combined with other forms. Small epidemics of exanthematic typhus are occasionally seen almost everywhere, and may usually, but not always, be traced to contagion.

Anatomical Appearances.—The characteristic exanthema of typhus, typhus roseola, cannot be recognized on the dead body any more than the exanthema of measles and scarlatina can, for the circumscribed hyperæmia, on which the roseola depends, disappears in the general paleness of the surface. Occasionally, indeed, this circumscribed cutaneous hyperæmia causes rupture of the vessels and small hæmorrhages in the cutis, and we then find the skin of the cadaver covered with more or less numerous and extensive petechiæ; but this is by no means constant, and we must not consider petechiæ as pathognomonic of "petechial typhus." The roseola-spots, observed on the skin of the patient during life (which we shall describe under the anatomical appearances, as we did measles, scarlatina, and small-pox), resemble those of measles, are about the same size, shape, and color; like these they unite to irregular figures, but, unlike the spots in measles, they are not capped by small papules. Sometimes they are on a level with the surrounding skin, sometimes they project slightly above it, while in abdominal typhus the eruption of roseola on the chest and abdomen is usually scanty and does not always exist; in exanthematic typhus it is not always confined to those parts, but usually covers the trunk and extremities in great numbers, and is so apparent that, even on superficial examination, it is not readily overlooked. The roseola rarely occurs on the face, and this one fact is sufficient to prevent a mistake in diagnosis between the exanthema of measles and typhus.

The other anatomical appearances also usually resemble those found after other exanthemata. If death takes place early, the bodies are but little emaciated, the rigor mortis is marked, and there is extensive hypostatic congestion in the dependent parts of the body. The muscles are dark-colored, the heart and great vessels contain cherry-colored blood. The bronchial mucous membrane is always strongly injected, and covered with tough mucus. In the lungs we find more or less extensive hepatization and atelectatic spots. The bronchial glands are swollen, but not infiltrated. There are no constant, marked changes in the intestinal canal and mesenteric glands.

The spleen is enlarged and soft. If death do not occur till late in the disease, the rigor mortis is less marked, and soon passes off; the bodies are more emaciated; the alæ nasi seem smoked, the teeth and gums are covered with sordes; the blood rarely contains any fibrinous clots; it is smeary, dark, or inclined to cherry-color, and the walls of the vessels are infiltrated. There is usually extensive hypostatic congestion in the lungs; the spleen is decidedly enlarged; it is sometimes the seat of hæmorrhagic infarction, or of small abscesses. There are no constant anomalies in the stomach or intestinal canal; at most, there is swelling of the solitary and *Peyer's* glands, such as is also seen in the acute exanthema. Nor do the other organs show any constant characteristic deviations from their normal state. In the rare cases, where death occurs still later from the sequelæ, on autopsy we find the most varied anomalies, suppurating parotid glands, the remains of croup and diphtheria, extensive necrosis of connective tissue, bed-sores, gangrene of the extremities, etc.

Symptoms and Course.—There are few diseases whose symptoms correspond so exactly in different cases, and which run such a similar course in different persons, as exanthematic typhus.

The period of incubation, which appears to last eight or nine days, rarely longer, is not usually free from symptoms of the disease; but these consist of slight chills, headache, disturbed sleep, loss of appetite, a feeling of dulness, depression, and discomfort; in short, of symptoms such as precede the outbreak of other diseases, and give no clew to the disease which is commencing. It is only during the prevalence of exanthematic typhus that we should suspect from these symptoms that the patient was infected with typhus poison; the suspicion would increase in certainty if there were, at the same time, catarrhal troubles, cough, coryza, burning feelings in the eyes, etc. The first two cases of exanthematic typhus that I saw in the Magdeburg hospital during the stage of incubation, I at first supposed to be cases of simple catarrhal fever. The intensity of these prodromatæ varies, so that, while they last, some patients go about their business, while others are confined to bed even at this stage of the disease.

The *stadium invasionis* begins with a single protracted chill of great violence, or with repeated slight rigors, followed by a continued feeling of great heat. After the first chill, the patients are rarely able to leave their bed; they feel excessively fatigued and weak, complain of heaviness and numbness in the head, occasionally also of headache, which is not unfrequently temporarily relieved by nose-bleed. These symptoms are followed by dizziness, flashes of light, tinnitus, deafness, pain in the muscles, trembling of the extremities on motion; the patients usually lie in a state of apathy, talk in their sleep, and even

while awake have a muttering delirium. Others are excited and restless, have anxious, wild fantasies, and can scarcely be kept in bed. At my first visit I found one of the patients, sent to me from jail with exanthematic typhus, in a strait-jacket. Besides these symptoms of disturbed innervation, there are almost always signs of intense catarrh during the invading stage; the eyes shun the light, and are red; there is an increased flow of tears; the nose is dry and stopped up, or its secretion is at first fluid, afterward more tenacious, and dries to crusts; the act of swallowing also is often difficult and painful, the tongue has a white coating, the taste is slimy; occasionally there are nausea and vomiting, and not unfrequently some diarrhœa. These symptoms are absent, or only slight, in some cases; a more constant one is a painful, hoarse cough, which brings up a tough mucus, occasionally mixed with blood. On auscultation we hear numerous ronchi. We have but few exact observations as to the grade and course of the fever; these are especially due to *Wunderlich*. According to these, even at first the temperature rises to 104° to 106°; the pulse is usually large, full, soft, rarely double, and beats about 100 in a minute. From the great loss of water from the body of the patient, induced by the increased perspiration due to the high temperature, the thirst is great, the urine scanty and of high specific gravity. Even in this stage, enlargement of the spleen may usually be discovered by percussion.

With the first appearance of the roseola spots, which occurs in the second half of the first week, usually between the third and fifth, rarely on the seventh day of the disease, the *stadium eruptionis et florescentiæ* begins (if we keep up the analogy between exanthematic typhus and the acute exanthemata, which, however, only exists at first, and is subsequently lost). At first the spots are few in number and only appear on the trunk, but they soon multiply and spread toward the neck and extremities, till finally the entire body, except the face, is covered with them; at some places they are more numerous than at others; the eruption is only exceptionally scanty; but even in these cases it is more extensive than the roseola eruption in abdominal typhus. The exanthema lasts longer than it does in measles or scarlatina; the roseola spots do not disappear till toward the end of the second week, when the fever defervesces and the other symptoms subside. The longer the roseola spots last, the more their bright-red color changes to a livid hue; they then disappear imperfectly under the pressure of the finger; part of them often change to petechiæ. The general symptoms do not improve with the outbreak of the eruption. It is true the patients complain less of pain in the head and limbs, but this is only because their mind is more affected; they can no longer think clearly, give slow and incomplete answers to questions,

and after their recovery they scarcely remember this period, during which they are in a quiet or noisy, violent delirium, and sometimes make constant attempts to jump out of bed and run away. They often become very hard of hearing. The tongue is dry and covered with a brownish crust; the cough has usually subsided, but the respiration is superficial and rapid; ronchi are more numerous, and at the dependent parts of the chest the percussion-sound is often dull. In some cases there is constipation, in others there is more or less diarrhœa, and the urine is often passed involuntarily. According to *Wunderlich*, in mild cases the fever retains till the end of the first week the height that it had attained the third or fouth day of the disease, or in the second half of the first week there is a slight decrease of the temperature, and on the seventh or eighth day a decided remission. In severe cases, on the contrary, the temperature increases during the second half of the first week, and there is no remission the seventh day. At this time the pulse is often very small and soft, and its frequence corresponds to some extent with the height of the temperature, so that in mild cases the pulse is about 100, in severe ones 120 or more, a minute. Toward the end of the first week even, the spleen is usually enlarged. In the second week, while the exanthema becomes more livid, all the symptoms increase, and about the middle of this week they attain their height. Then the patients lie almost constantly on their back, the eyes half closed, the knees rolling outward, the hands between the thighs, they are in a deep stupor from which they can scarcely be awakened. From time to time they mumble unintelligibly, make grimaces, gesticulate, pick the bedclothes, attempt to rise, and to thrust the feet out of bed, and it is evident that while all sense of shame and propriety is gone, and the real world is lost to the patients, they still live in an imaginary world, and their minds are more or less active. They do not appear thirsty, although the tongue is dry, and often covered with a blackish hard crust; but, if a glass of water be placed to their lips, they show great avidity for drink; attempts to satisfy their thirst often fail, because the tongue trembles or is stiff and immovable, and the act of swallowing is more difficult. The nostrils are usually filled with a sooty crust, the teeth and gums are covered with a smeary coating, whose decomposition causes a disagreeable fetor *ex ore*. In many patients the skin is at this time covered with petechiæ and miliary vesicles; in some there are symptoms of pneumonia and extensive atelectasis, in others those of parotitis. Not unfrequently the urine contains albumen. The above symptoms of severe constitutional disease and great disturbance of innervation, as well as those of pneumonia, collapse of the lung, parotitis, etc., occur even in those cases of exanthematic typhus that run a

favorable course, a fact that we should know, to avoid exaggerating the danger. Whether a remission occur on the seventh day or not, the fever always increases in the beginning of the second week. In mild cases, this increase only lasts a few days, and is not excessive; but in severe cases the increase lasts till the end of the second week, or even till the sixteenth or seventeenth day, and the temperature may become very high (108° or more). At this time the heart's action is not only much increased, but it is weakened; hence the heart-sounds are very imperfectly heard, the pulse is small and indistinct, the circulation is so much retarded that, in the extremities, the temperature of the skin approaches that of surrounding objects, and the hands and feet appear cool while the trunk is burning hot.

The third stage of exanthematic typhus, which authors very correctly call the *stadium criticum*, almost always begins in the latter part of the second week, or, exceptionally, in very severe cases, at the beginning of the third week. Before actually seeing such cases, it is almost impossible to imagine the wonderful change in the symptoms, during a single night, in the stadium criticum. In no other disease is there so rapid a change from an apparently hopeless to a very comfortable state. After a peculiarly marked exacerbation of all the symptoms, the patients fall into a quiet deep sleep for several hours, from which they awake with unclouded mind, but usually without any recollection of what they have passed through for the previous days or weeks. During this critical sleep the temperature often falls four degrees, the pulse twenty or thirty beats; the calor mordax disappears from the skin, and there is a gentle perspiration; the roseola spots become pale. In favorable cases defervescence is immediately succeeded by convalescence, which, however, is always slow. The patients sleep a great deal; on waking, their intellect gradually becomes less clouded, although they retain an idiotic, stupid expression for weeks. The dirty coating is thrown off from the teeth and gums, the tongue again becomes moist, and the appetite returns. Sputa cocta are raised by a loose cough; the skin, from which the roseola spots have disappeared, begins to peel off, any remaining petechiæ fade out, the temperature and pulse fall to the normal standard or below it, and the spleen decreases in size. But, even in the best cases, weeks pass before the patient can leave his bed and move about his room. In most of my cases the mental vigor returned even more slowly than the bodily strength.

The stadium criticum does not always pass immediately into convalescence; the typhus proper is often followed by sequelæ, which are apparently caused by the high fever, disturbance of respiration, continued abstinence, absorption of exudation, and other unknown causes. This view of the sequelæ of typhus is supported by the fact

that the same sequelæ also occur after abdominal typhus, puerperal fever, cholera, and some other severe diseases. Some patients, without our being able to find inflammations of any important organ or other material cause for it, are attacked by a new fever, of which they die, with the symptoms of great prostration, the already exhausted powers being rapidly consumed. In other patients, after the typhus has run its course, there are inflammation and suppuration of the parotids; in others, there are pneumonia, pleurisy, diptheritic and follicular inflammation of the intestine; in still others there are numerous furuncles, ecthyma pustules, or large abscesses in the subcutaneous and intermuscular connective tissue. In many cases, the slowly-healing bedsores prove dangerous, partly from the drain on the system, partly from reabsorption of ichor. Lastly, there is frequently thrombosis of the femoral veins, with its results. Recovery is by far the most frequent termination of exanthematic typhus in most epidemics, especially in those which are not very extensive. The circumstance that a fatal termination is less frequent than persons unacquainted with the disease would expect from the severity of the symptoms, is probably because the disease, which runs a cyclical course, is of comparatively short duration. The organism could not long stand such a fever as accompanies typhus. The hypothesis which we have frequently advanced, that the chief danger in infectious diseases is from the elevation of the temperature to a point where general paralysis is induced and the continuation of life impossible, is well supported by the observations made in *Wunderlich's* clinic during a small epidemic of typhus. In the fatal cases, death almost always resulted at the height of the fever; moreover, all patients in whom the temperature reached 108° died; and of fourteen patients that died, five, or more than one-third, had this high temperature. Of the patients whose temperature never rose above 105.5°, not one died. Besides the high temperature, capillary bronchitis, pneumonia, and atelectasis, are dangerous to the patient; other patients die of the sequelæ. Exhausting hæmorrhages, gangrene of the tip of the nose, fingers, toes, and lungs, and other complications, which formerly proved so destructive in the malignant form of exanthematic typhus, called putrid fever, are rarely observed in the present day; but, in the severe epidemics, such as occurred during the Crimean War, which cannot be compared in danger to those above mentioned, half the persons attacked died.

Lastly, I shall speak of a rarely-mentioned *abortive form* of typhus, of which I observed some cases in the Magdeburg hospital; a large number of observations made in the Prague epidemics of 1843 and 1848 exactly correspond with mine. The patients, who, in my cases, had always been exposed to typhus poison (among others, the nurse

of my assistant), complained of rigors, great depression, tightness of the head, frontal headache, weakness of the limbs, loss of appetite, and other symptoms common to the incubation stage of typhus. After a time there were stronger chills, with subsequent continued feeling of heat, increase of pulse, great apathy, and disturbed sleep. Some patients were slightly delirious. There were also catarrhal symptoms; the injected eyes were very sensitive to light, the nasal mucous membrane was swollen and dry, the nose stopped up; but, more especially, there was troublesome cough, by which a scanty tough secretion was brought up. We daily expected the eruption and the enlargement of the spleen, not doubting that the cases were commencing typhus; but these two characteristic symptoms did not occur; toward the end of the first week, the constitutional disturbance, fever, and catarrhal symptoms disappeared, and the patients began to convalesce; they recovered very slowly, and were not generally able to leave their bed before the end of the second week.

Treatment.—We have no remedy for arresting exanthematic typhus; hence we are limited to a symptomatic treatment. As this is to be directed chiefly against those symptoms that threaten the life of the patient, particularly against the fever, and since in treating abdominal typhus also combating the fever is the most important indication, we shall defer our remarks on this subject to the next chapter. We may also refer to the next chapter for the treatment of dangerous increase of respiratory difficulty, for great collapse, bed-sores, and other dangerous symptoms, for just the same points arise in abdominal typhus, and require the same remedies.

CHAPTER VIII.

ABDOMINAL TYPHUS—ILEOTYPHUS (TYPHOID FEVER).

Etiology.—The belief that exanthematic typhus is a simple, and abdominal typhus a complicated form of the same disease; that, in abdominal typhus, besides the changes that the blood undergoes in exanthematic typhus, there are changes in other organs directly concerned in the formation of the blood, in the intestinal and mesenteric glands, lacks all foundation. We shall not deny that there is a certain resemblance between the symptoms of the two diseases; but the resemblance is not perfect, and is not much greater than that between exanthematic typhus and typhous measles; hence, we do not consider it justifiable to regard the changes of the blood in the two diseases as being the same, or that the poison producing the one is the same as that causing the other. It is perfectly inadmissible to represent ab-

dominal typhus as a higher grade, exanthematic typhus as a lower grade of the infection with typhus poison, for there have been epidemics of the latter that were more malignant than those of abdominal typhus; and even in the milder epidemics of exanthematic typhus, the intensity of the symptoms, especially of the fever, was almost always greater in the individual cases than in most cases of abdominal typhus. While the fact that patients with varioloid often infect others with variola, and *vice versa*, shows us that variola poison and varioloid poison are identical; the fact that persons infected by patients with exanthematic typhus always have that disease, never abdominal typhus, shows us that the poison inducing the former is not identical with that of the latter, and that, in spite of some similarity of symptoms, the two diseases are of different nature. If the resemblance between measles and scarlatina were even greater than it actually is, the single fact, that infection with measles poison never induces scarlatina, would suffice to prove the non-identity of these diseases, and that the difference between them is not only one of degree; but we shall not deny the possibility that the two typhus poisons resemble each other more closely than do those of scarlatina and measles; nor shall we compare the prevalence of one or other form of typhus, at certain times and places, with the repeated epidemics of measles at certain times and places, or with the almost exclusive occurrence of scarlatina epidemics at other times and places. The belief in the relationship, or, to speak more generally, in the resemblance of the poisons which induce exanthematic and abdominal typhus, is supported by the similarity of the causes favoring their development, as well as by the similarity of their effects, that is, of the symptoms of the disease, as will appear from what follows.

I consider the extension by contagion, as well as by miasm, as even more probable in abdominal than in exanthematic typhus, or (to speak in a way that shall better show my position in regard to the views on contagion and miasm), that the germs which cause abdominal typhus may develop and multiply as well in the organism of a typhus patient as they would under favorable circumstances outside of it. It is true, I cannot prove the correctness of this hypothesis, but I must also deny that the recent assertions, that abdominal typhus spreads solely by contagion, have been proved, or even rendered very probable, by the facts adduced.

The *contagion* of abdominal typhus is not so intense as that of exanthematic typhus. There is no doubt that it clings particularly to the dejections of the patient, and that persons exposed to the emanations from typhus stools are most apt to be infected. On the other hand, I consider it doubtful if the contagion be transferred by the ex-

nalations from the skin and lungs (which, in exanthematic typhus, are certainly vehicles for the contagion, while in cholera they just as certainly are not so). Infection of the nurses and physician is rarely seen in abdominal typhus, and, when it occurs, it is always questionable whether it be due to the emanations from the patient, or from his dejections. At all events, using the bed-pans, night-stools, and privies, where the dejections of the patients have been emptied, appears more dangerous than being brought in contact with the patient himself.

The *miasmatic* origin of abdominal typhus is rendered probable by cases occurring in places removed from travel, where no cases of this disease have occurred for years, and where there is not the slightest suspicion of a contagious origin. If it be not considered an axiom that miasmatic diseases are never contagious, no wild hypotheses are necessary for the explanation of such cases. The most simple and probable explanation of them is, that the low organisms, which we suppose constitute the germs of abdominal typhus, may originate and increase not only in the bodies of patients and their dejections, but outside of them also. We at least partially know the circumstances that favor the origin and development of this poison, since we know that abdominal typhus occurs sporadically, and in so-called house-epidemics, especially in places where quantities of animal matter are decomposing. The absorption of the germs appears to take place chiefly through the lungs; but there are some instances where several persons, drinking water from a well that communicated with the privy, were attacked with abdominal typhus. I consider it doubtful whether the germs of this disease can also be swallowed with decomposing meat. The celebrated Andelfinger epidemic is often quoted as such an instance, but *Liebermeister*, in opposition to *Griesinger*, has shown that this was not abdominal typhus; while the latter, in opposition to the former, has shown that it was not a trichina epidemic.

In large, overcrowded cities, it can rarely be determined whether the cases of abdominal typhus be of miasmatic or contagious origin. At all events, the requirements for the development and increase either of spontaneous germs, or those from the dejections of typhus patients, are excessively favorable in such places, as the soil of large cities always contains quantities of decomposed and putrid animal substances. *Pettenkofer* reckoned that the excrement of Munich amounted to one hundred and fifty loads daily, and that on an average scarcely ten were removed, so that over seven-eighths of the excrement of the city remained and were absorbed by the soil. We shall hereafter show that the number of cases of intermittent fever in malarious regions varies with the rise and fall of the water in the marshes, because this induces the decay of vegetable matter, on which the production of the

morbific germ of intermittent depends. In the same way, experience shows that the number of typhus cases, in any place where the disease prevails, varies with the greater or less moisture of the soil; this is simply because certain degrees of moisture of the ground are favorable or unfavorable to the decomposition of animal substances on which the production of the typhus poison depends. It has often been observed that the sudden drying of previously moist soil, or, as is usually said with doubtful propriety, the sudden fall of the sub-soil water, corresponded with an increase of the typhus cases. The explanation of this is simple, according to what we have already said. But even the exceptions, where a moist state of the previously dry soil, sudden rise of the sub-soil water, coincided with an increase of the disease, are not surprising, nor are those where, while the height of the water remains the same, the number of cases varies. Of course other causes besides the moisture of the soil influence the decomposition of animal substances, and consequently affect the production of typhus germs.

The susceptibility to typhus poison varies greatly with the individual. There is one very interesting point, which is also seen in other infectious diseases, that persons who have lived for some time in a place peculiarly liable to the disease (as Munich), without being attacked, are in less danger when epidemics occur than those who have recently come to the place. It can scarcely be supposed that this is because the former were originally less disposed to the disease than the latter, but because, after a long absence from their native place, on their return to it, they are just as liable to the disease as new-comers who have only been there a short time. This fact probably depends on some unexplained accommodation to typhus poison. Statistics concerning the influence of age, sex, condition of life, and constitution, on the susceptibility for typhus, have shown that infants and old people are rarely attacked by abdominal typhus; persons of middle age are most liable to it; males are attacked oftener than females; strong, well-nourished persons, oftener than weak, badly-nourished ones; and that the disease is proportionately more frequent among the poor than among the well-to-do classes. Formerly, it was generally believed that tuberculous patients were never attacked by abdominal typhus; this is not absolutely true, although it rarely occurs. The same is true of patients with heart-disease, carcinoma, and other chronic or acute diseases, and of pregnant or nursing women. Pregnancy gives almost absolute immunity to the disease. Except in rare cases, one attack removes the susceptibility. Since 1820–1830, during which time exanthematic typhus has become more rare, abdominal typhus has become more frequent. With the exceptions mentioned in the preceding chapter, it is the common form of typhus throughout Europe.

It occurs far north in Russia and Denmark; in middle Europe, especially in Germany, France, and the Netherlands; and even in the south—in Italy, Syria, and Turkey—it is not rare. In Great Britain, exanthematic typhus is the common form, but abdominal typhus also occurs, especially in the country towns, and the parts of England not visited by Irish emigrants (*Hirsch*, *Griesinger*).

Anatomical Appearances.—It will be most convenient to speak first (as *Hamernik* does) of the *post-mortem* appearances when death has occurred early in the disease, before the typhus process proper has set in, and then to give a description of the lesions that are found when death has occurred later, during the repair of the changes induced by the typhus.

The bodies of persons who have died early in the disease do not appear greatly emaciated; rigor mortis is very marked; we find extensive hypostatic congestion in the dependent parts of the body, and occasional bed-sores commencing over the sacrum. The nostrils often appear smoky, and the teeth and gums are covered with a black coating. There are often numerous sudamina on the skin. On opening the body, the muscles appear very dark red, hard, and dry. The blood in the heart and large vessels is thick, dark colored, and contains little, loose, blackish-red coagula, rarely a small amount of discolored fibrin. Chemical and microscopical examinations of typhus blood have so far given negative results as regards the actual anomalies, that is, as to those which depend directly on the infection with typhus poison. The decrease of fibrin occurs in other infectious diseases also; the increase of blood-corpuscles, on which the dark color chiefly depends, seems to be only relative, and to result from the thickening of the blood, caused by great perspiration, and loss of water by diarrhœa. After the typhus has continued some time, the blood is consumed, and becomes poor in albumen and blood-corpuscles. The brain and spinal medulla show no constant changes corresponding to the severe functional disturbances of these organs during life. They sometimes contain more, sometimes less blood, and vary in consistence. We find changes in the respiratory organs in all cases; the typhous laryngeal ulcer already described (Vol. I.) is not unfrequently found, especially in certain epidemics. There are always signs of an extensive catarrh, even in the smallest bronchi, marked by dark redness of the mucous membrane, and scanty, tough secretion. At the dependent parts of the lungs there is more or less hypostatic congestion, sometimes only great hypostatic hyperæmia and condensation of the pulmonary tissue from swelling of the alveolar walls (splenization); sometimes hypostatic œdema; sometimes the so-called hypostatic pneumonia (Vol. I.). Besides the above, more or less extensive portions of lung are not unfrequently collapsed, or in

a state of atelectasis, from collections of secretion or swelling of the mucous membrane of the bronchi leading to them having rendered the passage impervious. In some cases also we find lobular and lobar pneumonia, not affecting the dependent part of the lung, and not depending on hypostasis, even at the height of the disease, although it is more common after it has run its course. The bronchial glands are swollen, vascular, and occasionally have a medullary appearance, such as we shall describe for the mesenteric glands. The heart is usually relaxed, its muscles pale, sometimes of a dirty-red color; the endocardium and lining membrane of the vessels are infiltrated, red, and discolored. The spleen is greatly enlarged, occasionally twice, or even six times its normal size; its capsule is tense, its parenchyma pulpy, and of a dark-violet or blackish-red color. In rare cases we find the capsule of the spleen ruptured, and blood poured through the rupture into the peritoneal sac. In the great curvature of the stomach, sometimes only the large vessels are distended, sometimes the mucous membrane appears dark red, from injection of the finer vessels, and relaxed from infiltration after death. The most important changes occur in the small intestine; to these ileotyphus owes its name. *Rokitansky*, on whose unsurpassed description of the "typhous affection of the mucous membrane of the ileum" we base our description, divides it into four stages. In the first or congestive stage, the mucous membrane of the small intestine is the seat of great hyperæmia. It appears swollen, relaxed, cloudy, covered with mucous and epithelial masses. This condition extends over all the membrane of the intestine, it is true, but it is most marked in the lower part, near the valvula *Bauhini*. The mesenteric glands are moderately swollen, soft, vascular, and dark colored. In the second stage, or that of typhous infiltration, the general redness and swelling of the mucous membrane increase, and concentrate on the parts around the solitary and *Peyer's* glands in the lower part of the ileum. In these tissues there are very peculiar changes, which are pathognomonic of typhus. More or less of the glands, and parts around them, swell so as to rise half a line or a line above the surrounding mucous membrane. These prominences are rather hard, and show through the mucous membrane with a gray or yellowish-red color; they have flat or steep edges; they are seated firmly on the muscular coat, and are intimately connected with the mucous membrane covering them. The size of the swollen solitary glands varies from that of a millet-seed to that of a pea. *Peyer's* patches, on the contrary, form patches from the size of a silver groschen to that of a dollar; they are generally oval in shape, and in the vicinity of the valve they usually coalesce, so that at this point they often cover a strip of intestine several inches long. On the cut surface

it looks as if the diseased intestinal glands were infiltrated with a soft, grayish-white, or pale-reddish encephaloid mass; and although it has of late been found that in typhus disease the intestinal glands are not infiltrated with amorphous exudation, but that there is an increase of their cellular elements which, even under normal circumstances, are peculiar; still the expression "medullary infiltration" has almost universally been preserved. Occasionally the degeneration extends beyond the follicles, and there is a "medullary infiltration" of the connective tissue of the mucous membrane in their vicinity, a cellular neoplasm, originating from the connective-tissue corpuscles (*Virchow*). In this stage the mesenteric glands are swollen to the size of a bean, or a hazel-nut, are of a grayish-red color, and quite hard. In the third stage, which *Rokitansky* calls the stage of relaxation, softening, and breaking down, the changes in the affected glands vary greatly in different cases. Not unfrequently the process becomes retrogressive, without the occurrence of destruction of the wall of the follicle or of the mucous membrane covering it; the swelling of the glands subsides, while their contents are reabsorbed after the cellular elements have been destroyed by fatty metamorphosis. These cases appear chiefly to correspond to the so-called abortive typhus. In other cases, the covering of the follicles is changed to a dry, friable slough, colored yellow by the fæces; this slough sometimes extends over the whole of the gland, so that its form and size correspond to those of the plaque; sometimes it is limited to part of the covering, and the slough has an irregular, angular, or roundish form. In still other cases, the individual glands composing *Peyer's* patches rupture, and empty their contents outwardly, without the covering sloughing. As a result of this, the surface of the plaque looks as if full of holes, or has a net-like appearance (*plaques à surface réticulée*). The mesenteric glands are most swollen in this stage; some of them attain the size of a pigeon's or even of a hen's egg. Their covering is usually bluish or brownish red, while their substance has a grayish-red, medullary appearance. In the fourth stage, or that of ulceration, the sloughs formed on the plaques or solitary glands are either thrown off in mass, or, after precedent disintegration, and a loss of substance, a typhous ulcer remains. *Rokitansky* gives the following as the most important characters of the typhus ulcer: According as it has resulted from a solitary follicle or from a *Peyer's* patch, it is round or oval, and, if there has only been a partial slough on the *Peyer's* patch, it is irregular; it varies in size from that of a hemp-seed or a pea to that of a dollar; its seat is in the lower part of the small intestine, and the ulcers proceeding from *Peyer's* patches are of course opposite to the insertion of the mesentery. The long diameter of the elliptical ulcer corresponds to the long axis of the

intestine; the margin of the ulcer is formed by a bluish-red, later slate-gray border of mucous membrane, about a line broad, which is movable over the surface of the ulcer. The floor of the ulcer is a delicate layer of submucous connective tissue, which covers the muscular coat. As soon as the slough is detached, the swelling of the mesenteric glands begins to subside, but they long remain larger and more vascular than normal. There are many deviations from this customary course of the disease on the mucous membrane; we shall briefly mention the more important of them: Occasionally, in the second and third stage, the hyperæmia of the mucous membrane over the swollen glands, and in their vicinity, becomes excessive. The mucous membrane is dark red, covered with ecchymoses; the patches resemble spongy, vascular, polypous proliferations, and the contents of the intestines are often mixed with a quantity of blood. A very serious event in the third, or sloughing stage, is the perforation of the intestine, which results from gangrene, not only of the mucous covering, but also of the corresponding part of the serous and muscular coats. The perforation is followed by severe peritonitis. Milder peritonitis also occurs without perforation. The deviations of the intestinal affection in typhus, as regards extent, are very marked; sometimes only a few *Peyer's* and solitary glands are affected; sometimes the ileum is almost covered with them. In the latter case we usually find the process further advanced in the vicinity of the valve than in the upper part of the intestine, and occasionally the difference of the stages in the different points is so marked as to lead us to suspect a succession of attacks. Not unfrequently the colon participates in the disease (colotyphus), then the solitary glands of the colon undergo the same changes as those of the small intestines. Far more rarely the process extends to the jejunum, and even to the pyloric portion of the stomach (gastrotyphus), the solitary glands, and certain portions of the mucous membrane, which usually correspond to the folds, undergoing the changes characteristic of typhus.

Where death occurs during the recovery of the ulcer and the other results of the disease, after the proper typhus process has run its course, the *post-mortem* appearances differ from those above described, and we must not limit ourselves to describing the processes which precede the healing of the ulcer, but must give a short glance at the state of the other organs, particularly as they show certain anatomical changes which never or very rarely occur during the first weeks of the disease. The bodies of patients who have died during the third or fourth week of the disease, or later, are more or less emaciated; the skin is pale, rigor mortis moderate, and, if there be decided anæmia, there is but little hypostatic congestion. In many cases the teeth and gums are

still covered with a blackish coating. Over the sacrum, trochanters, and elbows, we usually find bed-sores; there may be destruction of the skin only, or of the other soft parts also, extending to the bones. In many bodies there is slight œdema of the lower extremities, and, if one or other femoral vein be obstructed by a thrombus, there is very marked œdema of the corresponding limb. Lastly, on the skin we often find petechiæ, miliary vesicles, ecthyma pustules, and, in some cases, abscesses in the subcutaneous and intermuscular connective tissue, and suppuration of the parotid glands. On opening the body, the muscles no longer appear red and dry, but are pale and infiltrated. The blood in the heart and great vessels has lost its dark color, is fluid, and not unfrequently contains fibrinous clots, particularly when there has been inflammation of any organs. The brain is usually pale, moist, and the blood-points appearing on its cut surface are lighter, and contrast less with the white brain-substance than they did earlier in the disease. In the lungs, besides extensive hypostasis, we often find lobar or lobular pneumonia, and occasionally there are inflammatory exudations in the pleural sac; any laryngeal ulcers extend deeply and reach or destroy the perichondrium. In rare cases we find perichondritis laryngea without ulceration of the mucous membrane (Vol. I.). The heart is very relaxed and flabby, the endocardium and tunica intima of the vessels are greatly infiltrated. The swelling of the spleen has subsided, its capsule is often wrinkled, its tissue relaxed and pale; occasionally it contains hæmorrhagic infarctions. The distention of the vessels and capillary hyperæmia of the stomach have disappeared at the same time as the swelling of the spleen. The ulcers of the intestine are on the road to recovery, or are already cicatrized, especially in those cases where death was due to other causes. *Rokitansky* describes the healing and cicatrization of typhus ulcers as follows: The loose border of mucous membrane, forming the edge of the ulcer, becomes attached to the floor of the ulcer gradually from the periphery toward the centre, at the same time it becomes more pale and less thick; the delicate connective-tissue layer, which covers the muscular coat in the floor of the ulcer, becomes whitish, thickened, and is finally transformed into a serous plate, into which the adherent border passes imperceptibly, thinning as it approaches the centre. The mucous membrane gradually extends over this plate toward the centre of the ulcer, but, at the same time, becomes thinner, from the tension to which it is subjected. When the edges of the mucous membrane come together and adhere, the healing is complete. From the thinning of the mucous membrane, the cicatrix forms a slight depression; it is often somewhat pigmented, it is smoother than the parts around, and studded with a few tufts. Cicatrization of typhus ulcers never causes stricture of the intestines. As

the ulcers heal, the mesenteric glands return to their normal size, and often shrink to small, firm, slate-gray bodies. Some, also, become caseous and subsequently calcareous. Typhus ulcers do not always heal up, as above described. Occasionally the healing is delayed, but does finally occur. In other cases there is ulceration at the edges and base of the ulcer, which may cause erosion of vessels and abundant intestinal hæmorrhage or perforation of the intestine. It is difficult to determine whether rupture of the serous coat in these slow ulcers finally results from suppuration of its tissue, from extensive gangrene, or, after destruction of the muscular coat, from simple rupture of the thin wall. The fact, that, not unfrequently, errors of diet and mechanical causes, such as compression of the abdominal contents by vomiting, precede the perforation, seems to show that the perforation of the serous coat is often induced mechanically. When death occurs late in the disease, besides the remains of the typhus processes, we occasionally find the evidences of croupous and diphtheritic inflammation of the mucous membrane of the intestinal canal, especially of the large intestine, and very rarely in the gall-bladder also. Lastly, we must add that more or less extensive nephritis, as well as thrombi in the veins, is among the changes not unfrequently found after the typhus proper has run its course.

Symptoms and Course.—In many cases, the evident commencement of the disease is preceded for days or weeks by indefinite premonitory symptoms, which do not at the time enable us to determine the nature of the malady, but are of diagnostic importance after the disease has developed, as in doubtful cases they aid us in distinguishing typhoid fever from other affections that begin suddenly, without premonitory symptoms. These prodromata are feelings of general illness, mental disquiet, great dulness and relaxation, loss of appetite, indigestion, restless sleep disturbed by dreams, headache, dizziness, wandering pains in the limbs, which are usually considered rheumatic, and repeated epistaxis. These symptoms may last from a few days to several weeks.

We generally consider that the disease proper begins when the first chill occurs during the premonitory symptoms, or, if there has been no prodromal stage, when a chill notifies the patient of his illness. This chill is rarely so severe and continued as in intermittent fever or pneumonia; there is usually no shivering or chattering of the teeth. Frequently there is not a single chill, but several, and cases do occur without any chill. Hence, especially in inattentive patients, we cannot always tell whether the disease is at the seventh or eighth, or at the thirteenth or fourteenth day.

The pathologico-anatomical changes in the intestines during ty-

phoid fever do not correspond to any marked clinical stages, so as to enable us accurately to distinguish the symptoms corresponding to the stages of congestion, typhus infiltration, sloughing, and ulceration, and to give the symptoms of these different phases of the intestinal disease. Nor does the time that has elapsed since the first chill give any definite clew to the pathologico-anatomical stage of the bowel affection; but, on the other hand, we may suppose that, at the end of the third, or, at latest, at the end of the fourth week, the true typhus process has terminated, and that any existing morbid symptoms are only its remains, and belong to the secondary diseases of the blood and of different organs, induced by the typhus infection. Therefore *Hamernik* makes "a first and second period of the typhus blood-crasis;" in his communications from the *Pfeufer's* clinic, *Vogel* speaks of two groups of symptoms, the "intoxication" and the "reaction" symptoms; *Griesinger* makes a "first and second period of the disease." We also shall divide the symptoms into two classes, and shall speak first of the *symptoms of the disease itself*, afterward of the *symptoms of the sequelæ*. As we mentioned above, the former occur in the first three or four weeks of the disease, the latter later in its subsequent course.

Even during the first week the patient becomes very weak and much prostrated; but few can leave their beds during the first days; at the same time they complain of headache, which is chiefly in the forehead; of vague pains in the extremities, of buzzing in the ears, flashes before the eyes; of dizziness, which is particularly severe when they rise in bed, or attempt to walk. The sleep is restless and broken by dreams, in which the patients often speak single words, or whole sentences, in a loud voice. During the first week, while awake, the patient is usually perfectly conscious, but shows little interest in things around, and answers slowly and unwillingly to questions. There is great thirst, no appetite; there is a slimy or bitter taste in the mouth; many patients ask for an emetic, because their stomach is out of order. Occasionally there is diarrhœa, but at first the bowels are usually constipated, and it is not till the end of the week that the patient has several pulpy or even fluid stools daily; in still other cases, the bowels remain constipated throughout the week, and induce imprudent physicians to prescribe laxatives. If these or an emetic be given at the commencement of the disease, they almost always induce severe diarrhœa, which it is difficult to arrest. The passages are rarely accompanied by much pain. During the first week of typhoid, there is often repeated epistaxis, which is not apt to be abundant, and which relieves the headache. In most, but not in all cases, cough and mucous expectoration betray the bronchial catarrh,

which may be discovered by physical examination even during the first week. Among the objective symptoms comes next the changed appearance of the patient. While in bed, the face, especially the the cheeks, appears red; but, if he has been sitting up for some time, he looks pale and meagre. At first the tongue is rarely thickly coated, moist and broad, showing impressions of the teeth along its sides; it usually has a thin, whitish, epithelial coating, through which some papillæ project as red points; it is covered with a tough, slimy mucus, and appears narrow and pointed. The thin, epithelial coating usually falls off, gradually leaving, as *Vogel* aptly describes it, "a moist, red, smooth tongue, that looks as if covered with gold-beater's skin, or else is already inclined to dryness. If there be at first a thick, adherent coating on the tongue, it is usually detached from the point posteriorly, and from the sides toward the middle, so that the whitish yellow coating appears enclosed in a very red border, which constantly increases in width; but in some cases the detachment begins centrally, so that in the middle of the tongue there is a red stripe, that has a peculiar tendency to become dry, and at the sides two whitish-yellow, moist, slimy stripes. The central stripe is often broad anteriorly, and disappears posteriorly, so that on the point of the tongue we see a red triangle, with the apex posteriorly." In spite of this peculiar disturbance of nutrition on the surface of the tongue, and of the diminution of secretion in the mouth, on microscopical examination of the coating of the tongue, *Vogel* could find nothing characteristic. Palpation and percussion do not show any anomaly of the heart or lungs, but on auscultation even at first, and always by the end of the first week, we find a more or less extensive whistling sound (rhonchus sibilans), due to catarrh of the smaller bronchi. Even during the first days the abdomen is usually somewhat puffed up and tense; deep pressure over it is generally painful, and this sensitiveness to pressure exists not only in the ileo-cœcal region, but also about the navel and in the epigastrium. On pressure in the right iliac region, particularly if there has already been severe diarrhœa, we notice a gurgling sound, the so-called ileo-cœcal gurgling, whose diagnostic importance was formerly much overestimated. Toward the end of the first week, the enlargement of the spleen is ordinarily marked. The enlarged organ usually has a horizontal position; it rarely projects beyond the ribs, and is pressed upward and backward against the spinal column by the distended intestines. Hence, the typhus spleen is rarely to be reached on palpation, and, even when it can be touched, it is so soft that we are unable to define its outlines. But, if we lay the patient on his right side, with his left hand on his head, on percussing the lower ribs of the left side, we find a dull space, which may be six inches long by four wide, and which,

corresponding to the eighth, ninth, and tenth ribs, nearly reaches the spinal column posteriorly, and the margin of the ribs anteriorly. An increase or decrease of the spleen-dulness, to the extent of a centimetre or so, cannot be made out with certainty, in spite of what enthusiasts in physical diagnosis assert. Even when the dulness has undoubtedly increased or decreased slightly, we must bear in mind that this may depend not only on diminution or enlargement of the organ, but also on changes of its position. A greatly enlarged spleen may induce but little dulness when it is pressed into the hollow of the diaphragm by the distended intestines, and only a small part of it lies in contact with the thoracic wall. Toward the end of the first week, on careful examination, we often find a few pale-red, roseola spots, about the size of a lentil, on the epigastrium and neighboring portions of the abdomen and breast. Finally, the most important objective symptom is the fever. The temperature rises very regularly, in a manner almost pathognomonic of abdominal typhus. During the first week, the evening temperature is nearly two degrees higher than that of the morning; the morning temperature of the next day is about one degree lower than that of the preceding afternoon. For instance, a patient who has a temperature of 104°.6 this evening, will, as a rule, have a temperature of 103°.7 to-morrow morning, and to-morrow evening a temperature of 105°.5. Toward the end of the first week, occasionally there is no increase of evening temperature; but even then the morning temperature is almost always about one degree lower than that of the evening (*Wunderlich*). During the first week, the pulse usually increases to ninety or a hundred, or even higher. Its frequence does not by any means always correspond to the temperature of the body, for, besides this, there are other causes, not always evident, that influence the heart's action. Among other things, the pulse is accelerated from twenty to thirty beats in the minute, if the patient sits up in bed awhile, strains himself, or becomes excited in any way. Regarding the quality of the pulse, the blood-wave is usually quite large, but the artery remains soft during its diastole, and we often notice that the first pulsation is followed by a second, weaker one, that the pulse beats double. The double pulse, which does not occur exclusively in abdominal typhus, but is more frequent here than in any other disease, and consequently is not without diagnostic importance, is probably due to deficient activity of the contractile elements of the arterial walls, which are in a sub-paralytic state, as it is called. At least it may be shown that, if the arterial walls possessed no contractile filaments, but only elastic elements, the first pulsation, caused by the blood-wave, would be followed by a second evident pulsation ("*Nachschwingung*"). During the first week, in accord

ance with the severity of the fever, the urine is concentrated, high colored, and of increased specific gravity (1020 or more). If the patients replace the water they lose in perspiration and diarrhœa by drinking freely, the absolute amount of urine is not diminished (*Vogel*). The production of urea, which is increased in proportion to the supply of nourishment, as shown by *Vogel*, corresponds to the elevation of temperature, which depends on the increased transformation of tissue. Later in the disease, when the fever disappears, the increased production of urea also ceases, and, like the temperature, falls below the normal amount. The chlorides are diminished in the urine of typhus patients. The explanation of this is less simple than that of the increase of urea. It partly depends on less salt being eaten with the food, partly on increased excretion of the chlorides by the bowels, partly perhaps because, while the blood is deficient in albumen, it retains more salts. However, neither the increase of urea, nor the decrease of chlorides, nor, finally, the slight albuminuria, is characteristic of typhus, as it also occurs in other diseases accompanied by severe fever and copious exudations.

In the *second* week of the disease the complaints of pain in the head and limbs cease, but the dizziness becomes worse and the noises in the ears are accompanied by deafness, which, however, does not depend on disturbance of innervation, but, on a propagation of the typhous oral and pharyngeal catarrh to the Eustachian tube and cavity of the tympanum. The expression becomes more stupid, the inattention greater; the intellect, which was usually clear during the first week, becomes cloudy, and the patient gradually falls into a somnolent, stupid state, from which he can only be aroused with difficulty, and for a short time. In spite of the dry mouth, he manifests no desire for water, but drinks with avidity when a glass of water is placed to his lips. It is often necessary to urge the patient repeatedly, to make him show his tongue, and when he has at length, after several unsuccessful attempts, succeeded in protruding the trembling organ, he occasionally forgets to draw it back, and must be reminded and urged to do this act also. Toward the end of the second week particularly, the stools and urine are often passed in bed, because the patient does not perceive the necessity of evacuating the distended rectum or bladder, or because he neglects to contract the sphincters energetically by an action of his will. Many patients lie unconsciously on their backs; if placed on the side, the body and limbs follow their own weight, without the patient making any attempt to change his position, even if it be uncomfortable. The occasional trembling movements of the lips, or a few incomprehensible words that the patients mumble, alone show that the mental activity is not all lost (febris nervosa stupida) Other

patients, who are just as insensitive to the external world, who denude themselves without any modesty, do not answer questions, and scarcely react to the strongest irritants, show by their whole manner that they are living in an excited dream; they are constantly agitated, throw off the bed-covers as fast as they are replaced; thrust first one foot, then the other, out of bed, attempt to stand up or run away, talk loud or disjointed words, gesticulate, speak unknown tongues, and become morose and angry when they are held or in any way restrained (febris nervosa versatilis). It is astonishing what energy and persistence such patients often show in attempting to carry out their morbid impulses. Occasionally, during the entire disease, a certain circle of hallucinations recurs; the patients pursue any object that they cannot attain, are grieved by any sorrow that they are constantly combating, etc.; in other cases the phantasms alternate confusedly, with others having apparently no connection with them. Occasionally this alternation is so great that during the day the case appears to be a febris nervosa stupida, while during the night it seems to be a febris nervosa versatilis. In some cases the patients are constipated, even during the second week of typhoid fever; but, as a rule at this time, there are several watery evacuations daily. They vary in number from three to six, or even to twenty or more in twenty-four hours, but we cannot, from their frequence, decide the number and extent of the typhus ulcers, as the diarrhœa does not depend on them, but on the catarrh accompanying them; it is exceptional for the stools to be excessively frequent, and it is most common to have only three or four passages daily. The dejections have the color and appearance of badly-cooked pea-soup, in which the meal is not thoroughly mixed, but has sunk to the bottom. They have an alkaline reaction, contain only traces of albumen, and have no microscopic elements or chemical constituents peculiar to typhus. The upper watery layer contains chiefly salts, and owes its alkaline reaction to the large amount of carbonate of ammonia in it. The lower stratum consists of remains of food, detritus, epithelium, mucous corpuscles, numerous crystals of triple phosphates, and small yellow flocculi and globules, of whose origin and character we know nothing definite. Respiration is hastened and superficial. In some cases, in spite of the extensive catarrh, there is neither cough nor expectoration; in other cases the patients cough a good deal, and expectorate quantities of tough mucus. The objective symptoms also have changed to some extent in the second week. The cheeks are now more brownish red or bluish, the eyelids are half-closed, there is dried mucus at their corners, the conjunctiva is injected, the nostrils smoky-looking. A brown, tough mucous, fuliginous coating clings to the teeth and gums, and the tongue is covered with a brown

crust, which gradually becomes blackish by admixture with blood from small fissures in its mucous membrane. Decomposition of the coating of the tongue and teeth causes a very disagreeable, penetrating odor; movement of the tongue is much impaired, so that speech becomes indistinct, chewing of hard substances often impossible, and even drinking difficult. Physical examination of the thorax almost always shows more or less extensive condensation of the dependent portions of the lungs; on both sides of the spine the percussion-sound is less intense; on auscultating over the back, we find a weak vesicular or undecided breathing and fine mucous râle, rarely bronchial respiration, and at other parts of the chest there are numerous loud rhonchi. The belly is usually puffed up by the excessive meteorism of the intestines, which has never been explained. Its sensitiveness to pressure continues, as does the ileo-cæcal gurgling in many cases. The enlargement of the spleen has increased; and that organ is pressed still farther backward and upward by the inflated intestines. In some cases the roseola spots have become quite numerous, and have spread from the epigastrium and lower half of the thorax to the back; sudamina frequently occur. Among the objective symptoms of fever, the bodily temperature in the evening is usually 104° to 106°; in the morning there is only a slight remission. The pulse, which is less full, soft, almost always double, is often as high as 110 to 120, or more. In many cases the urine contains traces of albumen.

In the *third* week of typhoid fever, the patient becomes excessively weak; he cannot sit up; and, if the bed be inclined, he slides down toward the foot of it, as often as he is lifted up. In the relaxed muscles we may often see and feel contractions of single fasciculi, the so-called subsultus tendinum. The somnolence and stupor reach the highest grade; the noisy delirium ceases, and the restlessness gives place to increasing stupor; some patients make automatic motions with the hands, or pick at the bedclothes, and almost always pass their fæces and urine in bed. Occasionally the detrusor urinæ is also paralyzed, and the bladder excessively distended. The coating on the tongue and teeth becomes thicker, crusty, and fetid; the stammering speech grows unintelligible; drinking is very difficult. The chest and abdominal symptoms, also the frequency of respiration, rhonchi and mucous râles, dulness along the back, diarrhœa, and meteorism, become very decided. The spleen does not enlarge any more, but its swelling begins to subside. In the third week the roseola spots also begin to grow pale, while the sudamina increase, and petechiæ sometimes appear. In most patients there is erythema about the sacrum and detachment of the epidermis, and the exposed cutis sloughs. At the commencement of the third week there is also apt to be an in-

crease of the temperature and of the pulse. The morning remissions are indistinct. Fatal termination is most common in the third week, if not induced by some peculiar accident, death results from œdema of the lungs, after the prostration, weakness, temperature, and pulse have reached the highest grade. The more the respiration is affected, the sooner and more readily paralysis of the heart occurs. In favorable cases there is a subsidence of the symptoms about the middle of the third week. The state of deep sopor, during which the patient lives a dream-life, gives place to natural sleep. While awake, the expression of the patient shows that he pays some attention to the things about him, which, at the height of the disease, had no existence for him; he also recognizes his attendants. The first glance in which affection and gratitude again appear may be regarded as an advance, although the danger is not yet over, and although the hopes awakened by this and other signs are often blasted. The more quiet and continued the sleep, the clearer the intellect usually is during the waking state. The patients begin to complain of the bed-sores, and to lie on the side, so as to avoid pressure on the sore parts. They no longer pass their urine and fæces in bed, but call for the bed-pan when they wish to have a stool, or empty the bladder. The number of respirations decreases, the patients cough oftener, and more strongly, and easily expectorate the mucus collecting in the bronchi, which has become less tough, and is usually yellowish. The passages are less frequent, and contain some consistent matter. The blue, sodden appearance of the patient disappears, the face becomes paler. The tongue grows moist at the edges and tip, its coating is gradually thrown off; speech becomes more intelligible; there is less difficulty in drinking. We hear moist râles in the chest, the dulness along the spine disappears; even at these points the respiratory murmur becomes distinct, meteorism decreases, the spleen dulness grows less, the roseola spots disappear. With the abatement of the other symptoms, the difference between the morning and evening temperatures becomes remarkable; while the thermometer placed in the axilla still rises to 104° to 106° in the evening, in the morning it is often only 101° to 103°, or even less. These lower degrees are not observed in the evening for some time yet. As the temperature decreases, the pulse also falls, although the two do not keep pace. At the same time the pulse becomes fuller, and loses its rebounding character. This general improvement, which often does not occur till the *fourth* week, while the symptoms have maintained their intensity, or even increased till the end of the third week, often passes directly into convalescence, and the slow recovery alone shows that remains of the typhus process, especially intestinal ulcers, still exist; in other cases, the improvement is only temporary;

the symptoms grow worse, and the patients die of paralysis of the heart and suffocation. In some cases, finally, the above symptoms are followed by those of retarded repair of the typhous local affections and sequelæ. The appetite, which often becomes wolfish during convalescence, may prove fatal, if the physician be careless, or the patient intractable. We shall again refer to the danger accompanying this symptom. Almost all convalescents lose their hair. But, as the hair-follicles have undergone no permanent disturbance of nutrition, a new growth of hair sprouts up soon after the old has fallen out.

We have attempted to give as comprehensive a view as possible, and, at the same time, a tolerably complete one, of a severe "normal" case of typhus.* But it would lead us too far to describe with equal fulness the numerous and varied deviations that it presents in different cases, and we must satisfy ourselves with noting the most impor-

* *Wunderlich*, who has made a very large number of measurements of temperature in typhoid fever, gives the most important results of his observations about as follows: The course of the fever is typical and perfectly characteristic; it distinguishes abdominal typhus from any other disease. Besides the cases which exactly follow the regular type, there are others which deviate from it—irregular cases. The causes of the irregularities cannot be recognized in every instance. The course of the disease shows two sharply-bounded, distinct periods, which correspond to the deposition and reabsorption of the infiltrations and exudations. These periods agree with certain portions of time, and in regular or nearly regular cases they respectively correspond to the first and last half of the disease. In mild forms, the first period lasts only a week, or a week and a half; the entire disease lasts three or four weeks. In severe cases, the first period lasts two, three, or three and a half weeks; the entire disease lasts five, six, or occasionally from eight to ten weeks.

In the *first week*, it is such an absolute rule for the temperature to rise two degrees toward evening, and to fall one degree before the next morning, that, if the temperature on the second or third day be 104° or over, we may exclude typhoid; the same is true, if between the fourth and sixth days the evening temperature do not rise to 103°, and, lastly, if the evening temperature begins to decrease again as early as in the second half of the first week. A decided increase of temperature during the first week is generally an unfavorable sign, while a slight increase is favorable.

In the *second week* we may exclude typhoid, if the temperature be below 104° on one or more evenings between the eighth and eleventh day, and, conversely, on the evenings of the second week, scarcely any other acute disease shows repeated rise of the evening temperature to 104°. A favorable course of the disease during the second week renders it probable that the third week will be still milder, while, on the contrary, a severe and unfavorable second week may lead us to expect that the subsequent course of the disease will be unfavorable. Among the favorable indications during the second week are: an evening temperature between 104° and 105°; a morning temperature one or two degrees lower; late occurrence of the exacerbation (not before 10 A. M.); early occurrence of the remission (before midnight); regular and daily moderate decrease of temperature, as compared with that of the preceding day. Among the unfavorable indications are: continued elevation of the morning temperature; increase of the evening temperature to 105°.5, or more; early occurrence of the daily

tant modifications of its course, and the severest and most dangerous accidents that present themselves.

First, there are many cases which are not characterized by any remarkable lack of intensity of the symptoms, or by any other peculiarities during the first week; but in the second week the symptoms do not grow worse and prove dangerous, as in "normal" typhus, but they decrease, and toward the end of the second or third week have all disappeared. *Lebert* has proposed the name *abortive typhus* for these cases, and it seems to me preferable to other names, such as "febricula," "febris typhoides," etc., because it better expresses the fact that these cases are only modified, benign, brief forms of typhoid, and not a peculiar variety of disease. After what we have said, it would be a useless repetition to give a description of the course of abortive typhoid during the first week; so we shall only remark, that many of the cases which former writers call "gastric fever" or "mucous fever" are to be regarded as abortive typhus. The old customary precaution, of waiting till the ninth day of the disease before saying whether the case was "one of gastric or nervous fever," was and still is very justifiable. The designations "gastric fever" and "nervous fever," in common use, exactly correspond to what modern physicians mean by "abortive typhus" and "normal typhoid," and since the laity do not understand precise scientific terms, it would be well to keep up this old custom. As in the first week the thermometer is the most certain means of distinguishing typhoid from a genuine febrile gastric and intestinal catarrh (Vol. I.), so in the second week it is the most certain means of deciding whether the disease will be an abortive typhus or not; the other symptoms are far more deceptive. If we find that on the eighth or ninth day of the disease the temperature ceases to rise, but gradually falls, and especially if we find decided morning remissions at this time, we may be almost certain that the case is one of abortive typhus. Cases where the temperature rises again

exacerbations; late occurrence of the daily remissions; and very high temperature at any time.

In the *third week*, in mild cases, there are great morning remissions, so that the morning temperature may be three to three and a half degrees lower than the evening temperature, and may become normal toward the end of the week. And from the middle of the week the evening temperature also decreases. In severe cases, on the other hand, the temperature sinks but little during the third week, or else it maintains the same height, or even rises. In the latter case, we may almost positively expect a severe fourth week, and not look for a decided decrease of the fever before the fifth week.

We may look for the approach of death, if the temperature remain for some time at 107°; if it suddenly rise to 107°.5 or 108°, or when it suddenly falls very low, say to 94°. We must not expect perfect convalescence till the temperature is normal in the evening also.

toward the end of the second week, and the subsequent course of the disease is as above described, are rare. Even in abortive typhus, the sleep is restless and disturbed by dreams, and patients talk in their sleep during the second week; but when awake their mind is clear, and only a more or less marked apathy reminds us of the coma vigil of severe cases. The bronchitic symptoms are moderate; the daily evacuations are few in number, or there is no diarrhœa. The patient looks pale and worried rather than blue and sodden. The tongue is inclined to dryness, but is not covered with a firm crust. There is either no deafness, or else it is only slight, like the oral and pharyngeal catarrh. The abdomen is soft and only moderately prominent. Frequently there is no ileo-cæcal gurgling or sensitiveness of the abdomen to pressure. The spleen-dulness is but little increased. Only exceptionally a few roseola spots occur on the epigastrium. During the morning, and while the patient remains in bed, the pulse is only moderately hastened. In the third week, or toward the end of the second, the temperature is usually normal, and is only elevated moderately toward evening; the tongue remains moist, and the patient begins to have some appetite. The morbid symptoms of the thoracic and abdominal organs have disappeared, particularly the diarrhœa. Many patients are anxious to get up; but, on attempting to rise or move about the room, are usually surprised at their feebleness, of which they were not aware while in bed. The strength returns slowly, and this tardy convalescence shows the severity of the disease that the patient has had. The conjecture above advanced, that in abortive typhus there is no sloughing of the diseased intestinal glands, is not founded on *post-mortem* examination, for which the favorable course of the disease gives no opportunity, but on the early cessation of the diarrhœa, and the absence of the sequelæ, so common in severe and tedious cases of typhus, especially of those depending on retarded cicatrization of the intestinal ulcers, and on the ulceration occurring in their place.

There is a second modification of typhus which differs materially from that above described; it is usually termed *typhus ambulatorius*. It is not very rare for persons who have only suffered from a slight amount of weariness and depression, loss of appetite and slight diarrhœa, but have still been able to attend to business, or go on a journey, to die suddenly of perforation of the bowel or intestinal hæmorrhage. On autopsy of such cases, we may find numerous intestinal ulcers along with sloughing patches and medullary infiltration of the mesenteric glands; in short, the anatomical changes of advanced typhous disease of the intestines. It is difficult to explain this peculiar form of disease, except on the supposition that, under some circum-

stances, the infection with typhous poison may give rise to local changes in the intestines, but induce so little alteration in the blood and nutrition as to cause no noticeable functional disturbance.

While in typhus ambulatorius the constitutional disturbance and fever are so slight as to be scarcely observable, some other cases of typhoid are characterized by unusual intensity of the constitutional disturbance, and especially by the height of the fever. In these cases the disease usually runs a "tumultuous" course. Even in the first week the temperature rises to 106° or more, and the pulse beats 120 to 130 per minute. During the day, the patients lie in a state of deep somnolence, and look stupid; at night they have high delirium, and can scarcely be kept in bed. Subsultus tendinum, picking at the bedclothes, and occasionally convulsive movements, begin early. The intensity of the local symptoms usually corresponds to that of the general disease. The tongue becomes dry and crusted early; even during the first week the bronchitic symptoms, the signs of condensation of the lungs, the meteorism, diarrhœa, and the enlargement of the spleen, attain as high a grade as they usually do by the end of the second week. Toward the end of the first or the beginning of the second week the patient is greatly prostrated, slips down in bed, has a small, irregular pulse, and very rapid superficial respiration. Such patients often die early of paralysis of the heart and œdema of the lungs. In other patients, after a tumultuous course during the first week, the symptoms moderate and show no marked peculiarities during the second or third week. Between typhus of great intensity and tumultuous course, and typhus of moderate intensity, and between this and typhus ambulatorius, there are numerous intermediate forms, which we shall not fully describe. Many cases, treated for weeks as "gastric" or "mucous" fever, and finally recovering without any affection of the intellect or dryness of the tongue, are mild cases of typhoid; but it is certainly going too far to scratch gastric and mucous fevers entirely out of the list of diseases. As we have shown above, the height of the bodily temperature is more important than the roseola or the enlargement of the spleen in diagnosing between typhoid, that is, an infectious disease, and a genuine intestinal catarrh.

The appearances are also modified when the typhous intestinal disease is very slight, or, to use the customary expression, when the typhus localizes itself very slightly, if at all, in the intestines, and "runs almost exclusively in the blood." Since, in the first weeks especially, the distinction between abdominal typhus and other acute infectious diseases is based chiefly on the intestinal symptoms, an exact diagnosis is entirely impossible in such cases, and we must occasionally content ourselves with a probable diagnosis, by the exclusion of other

infectious diseases. If the intestinal symptoms fail entirely, or if there are only slight indications of them, while the bronchitis is severe and is early complicated with collapse and hypostasis of the lung or with decided pneumonia, we have the disease which, especially in those fatal cases where autopsy has shown medullary infiltration of the bronchial glands, has been called pneumo-typhus or broncho-typhus. It is evident that these cases, where the patients have a very cyanotic look and hastened and painful respiration, belong to the malignant forms. While the severe fever increases the production of carbonic acid, the disease of the bronchi and lungs impedes the excretion of the extensively formed deleterious gases. The formation of typhous ulcers in the larynx is not usually accompanied by any characteristic symptoms, and modifies the appearance of the disease so little that we are less justified in making "laryngotyphus" a separate class than we should be in so considering broncho- and pneumo-typhus. But while, during the first weeks of the disease, laryngeal ulcer is not usually recognized, and is accidentally found on autopsy, it plays a very important part in the sequelæ of typhus.

Among the accidents that interrupt the normal course of the disease during the first weeks, the most important are perforation of the bowel, intestinal hæmorrhage, and the abundant epistaxis which occasionally occurs during the second or third week.

The perforations of the intestine, which occur during the first weeks of the disease from the formation of a slough not only in the mucous coat covering the patches, but also in the muscular and serous coats at the affected parts, induce intense peritonitis; but since, previous to the perforation, there has occasionally been adhesion between different loops of intestine, from inflammation of the peritonæum, this inflammation is sometimes partial, not total, at first. The first signs of perforation are a severe pain in the abdomen, which usually arouses the patient even from deep stupor, and which becomes excessive on the lightest pressure over the abdomen; the patient collapses suddenly, the face becomes distorted, the pulse small, the extremities cool, and death usually occurs in from twenty-four to thirty-six hours, with the symptoms that we have described (Vol. I.) as indicative of peritonitis from perforation of the bowel. The most certain point in the diagnosis of perforation of the intestine is the escape of air into the peritoneal cavity, which is shown by the liver being pressed away from the abdominal wall and a consequent disappearance of the liver-dulness. If this symptom be absent, there is at least a possibility that the peritonitis is not due to perforation of the intestine.

Intestinal hæmorrhages, occurring in the first weeks of typhus, from erosion of the vessels on the detachment of the slough, or from rupture

of the overfilled capillaries in the spongy elevations of the mucous membrane over the plaques above described, if abundant, often betray themselves before the appearance of blood in the dejections, by the collapse of the patient, which is accompanied by a sudden fall of his temperature, and occasionally by a clearing of the intellect. The loss of blood is often very considerable, but patients rarely die as a direct result of the hæmorrhage; the bleeding usually ceases, the typhus pursues its course, but the strength of the patient does not carry him through, and most patients die, sooner or later, after the hæmorrhage, from the exhaustion that is completed by the fever and diarrhœa.

The abundant hæmorrhages from the nose during the second and third weeks are far less dangerous than the intestinal hæmorrhages; they are due to an acute hæmorrhagic diathesis, which frequently occurs from excessive debility. They may be so severe as to require the tampon, they exhaust the patient, and at least retard convalescence. In women there is often hæmorrhage from the genitals also, which does not always occur at the menstrual period. Although the loss of blood is not great, those hæmorrhages which are greeted by the laity as favorable signs are usually *mali ominis*.

If we bear in mind that most typhus patients recover very slowly even when the intestinal ulcers heal, the fever disappears, and the appetite returns immediately after the disease has run its course, and do not fully regain their strength for five or six weeks, it is not difficult to understand that *retarded healing of the intestinal ulcers*, and the continuance of even a moderate fever by the bowel-disease, should increase the danger to the patient. In these cases we find the typhus proper followed for weeks by symptoms of an asthenic fever, or of a fever in debilitated and anæmic persons. The intellect remains clouded, although the violent delirium usually disappears; the patient becomes weaker, and slips down in bed more frequently than before. The tongue does not again become moist, or else becomes dry again, the bronchitic symptoms disappear, but the hypostases extend; enlargement of the spleen and roseola spots can no longer be found, but the meteorism and diarrhœa continue more or less marked. The bed-sore over the sacrum spreads, increases in depth, and may cause frightful destruction. Bed-sores also develop over the trochanters, elbows, and on the knees also, if the patients be laid on the belly. Petechiæ and ecchymoses form at different parts of the body, especially where there has been any temporary pressure. Emaciation becomes excessive; the pallid skin is constantly bathed in perspiration. The visible mucous membranes also become pale and bloodless. There is often slight œdema of the lower extremities, or excessive œdema of one leg from thrombosis of the femoral vein. Many patients die in the sixth

or seventh week, or even later, of this slow form of typhoid. In other cases the diarrhœa ceases, the intestinal ulcers heal; but the bed-sores induce fatal exhaustion; at least, we not unfrequently find deep destruction of the soft parts and exposure of the bones, and recently-healed intestinal ulcers, as the only *post-mortem* appearances in cases that have proved fatal late in the disease. The pneumonias, pleurisies, suppurations of the parotid, diphtheritic inflammations of the intestines, nephritis, and other sequelæ of typhoid above mentioned, show themselves, just as in other debilitated persons, by objective rather than by subjective symptoms. Chills, and renewed increase of the bodily temperature, should excite the suspicion of one of these consecutive diseases and induce a more careful examination. Repeated chills, very high temperature, quick collapse, generally depend on pyæmia, induced by absorption of ichor from the bed-sores. The occurrence of severe pain in the larynx, hoarseness, aphonia, and the signs of acute laryngeal obstruction, indicate perichondritis laryngea, which is caused by typhous laryngeal ulcers penetrating deeply, but also occurs independently of this as a sequel of typhus.

Lastly, we must mention that in some cases perforation of the intestine occurs in the fifth or sixth week, not only while the patient is debilitated by the fever, induced by sluggish ulcers, but even while convalescence is going on perfectly well. Far more rarely hæmorrhages occur at this time, from slowly-healing ulcers.

Recovery is the most frequent termination of typhoid fever; it takes place in about three-fourths of all cases; but some epidemics are far more malignant, while in others the mortality is much less. In most fatal cases death occurs in the second or third week of the disease, that is, at its height; but we have already stated that, where the course is very rapid, the disease may prove fatal in the first week, and, in protracted cases, not till the fifth or sixth week, or even later. The different causes of death have been sufficiently described when speaking of the symptoms and course.

Sometimes typhoid ends in incomplete recovery; sequelæ, such as disturbances of innervation, neuralgias, partial paralyses, partial anæsthesias, or mental disturbances, remain; occasionally typhus leaves a tabes and a lasting anæmia and hydræmia, which are not thoroughly understood. The material changes on which these disturbances of innervation depend have not been discovered on *post-mortem* examination. The supposition, that the remaining sickness and the deficient formation of blood depend on destruction of the intestinal glands and impermeability of the mesenteric glands, is incorrect. Pulmonary consumption not unfrequently develops during convalescence from severe typhoid fever.

TREATMENT.—Prophylaxis requires that, in large cities, where typhoid fever is endemic, there should be sanitary police rules to prevent the soil from becoming soaked with putrid decomposing materials. Of late, many attempts have been made, by drainage in cities, to remove the moisture of the soil, which greatly favors the decomposition of animal substances, and, consequently, the occurrence of typhus and cholera germs. Should the drainage succeed, and should it decrease the number of typhus and cholera cases, as the drying up of swamps sometimes does that of intermittent fever, the still youthful science of hygiene would boast of a brilliant triumph. Some physicians still doubt the contagiousness of abdominal typhus, or, at least, consider it as not proved. But such views must not be brought to the bedside of the patient; here the fact, that the non-contagiousness has not been proved, must make us act as if its contagiousness had been demonstrated. The sick should be isolated from the well, and only those attendants required in caring for the patients should be allowed in his vicinity. It is just as important that all healthy persons, who can be spared, should be removed from the locality where the infection of one or more persons has occurred. I have learned of one very sorrowful instance in Rheinland during the past few years, where, apparently, from the neglect of this precaution, almost all the members of one family, one after another, died of abdominal typhus. Lastly we must take care that the dejections of patients be not thrown into privies used by other persons. We know no prophylactic remedies that protect from infection by typhoid.

When abdominal typhus has once begun, we may, under some circumstances, attempt to cut short its course. It is now generally recognized that this cannot be done by emetics or venesection, which, for a time, were recommended for this purpose; in fact, these measures almost always have an injurious influence on the course of the disease. Only when there is undigested food in the stomach, should we give a dose of ipecac.; in all other cases we must remain immovable to the senseless and often annoying requests of the laity for the administration of an emetic. The case is very different with a few large doses of calomel. After the accurate observations of *Wunderlich*, we can scarcely doubt that by this remedy we may, in some few cases, cut short the disease (according to *Wunderlich*, one or two five-grain doses are enough), and that in the great majority of cases where this remedy is given during the first week, and before the occurrence of much diarrhœa, the course of the disease is rendered milder and shorter. The experience of *Pfeufer's* clinic, as well as my own, perfectly agrees with *Wunderlich's*. We shall not attempt to say whether the calomel has a favorable influence on the typhous intestinal disease, by op-

posing the sloughing and ulceration, and whether, consequently, we can only expect benefit from it in the first weeks of the disease, when these changes have not yet taken place. *Willebrand* has lately recommended iodine as a specific in typhoid, and the results published by him, *Liebermeister*, and others, urge us to make more trials with the preparations of iodine in this disease, although the treatment is not a new one. *Willebrand* dissolves six grains of iodine and twelve of iodide of potassium in one drachm of water, and gives three or four drops of this solution in a wineglassful of water every two hours. It is said that, even after one, two, or three days of this treatment, there is a decided remission of the temperature; sordes do not come on the gums, or they disappear early; the fever ceases in an unusually short time.

In most cases of typhus, the indications are to protect the patients from all injurious influences, to combat dangerous symptoms, and to maintain the strength of the patient by dietetic rules. In the first place, wherever circumstances permit, we should see that the sick-room be not too small, and be very careful to have it well ventilated. Fresh, pure air is very necessary for all typhus patients, and they do not catch cold nearly so readily as the laity imagine. The temperature of the room should be regulated by the thermometer, and should be kept as nearly as possible at 60° to 65°. In North Germany there is a belief that a vessel of water under the bed prevents bed-sores. If it do not fulfil the expected end, this practice is probably useful in maintaining a certain degree of moisture in the room. We should also attend carefully to the patient's bed. The coverings should not be too heavy, and the sheet should have no creases in it. The bed and body linen should be changed as often as soiled. We should not neglect to see that the body of the patient is kept scrupulously clean throughout the attack. Even among the neatest persons this is not superfluous, for they are often prejudiced, or else fear to injure the patient by exposure, or do not carefully inspect the parts about the anus and sexual organs, so as to cleanse them from any excretions. Neglect of this precaution frequently cannot be subsequently made good. The most insignificant erythema on the nates, which might have been avoided, may prove very dangerous late in the disease, by forming the commencement of an extensive bed-sore. If the patient be too weak to clean his own mouth, we should have it washed out regularly with a linen rag wet with cold water, or, still better, with cold soda-water. Particular care should be paid to washing off the sticky mucus clinging to the teeth and gums, before it dries and decomposes. Even when the patient is in a state of stupor, he usually shows some signs of pleasure and gratitude after this proceed

.ng. The best article of drink is pure spring-water or soda-water; if there be severe diarrhœa, we may give oat or barley water. All additions of fruit-juices, vegetable acids, toasted bread, etc., to the drinks, soon become objectionable to the patient. The patient must drink freely, in order to replace the loss of water induced by the copious perspiration. If, during the advanced stages of typhoid, they do not ask for drink, because they do not perceive the want of it, it should be offered to them. Badly-instructed or thoughtless nurses sin a great deal against this rule. Shall we give the patient nourishment, or place him on absolute diet? Views vary greatly on this point. Most German and French physicians consider the administration of meat-broths, eggs, and other nutritious substances in fevers generally, as so decidedly injurious, that they regard "fever-diet" and water-soup as identical. From England, there is an accusation against the German physicians particularly, that their dietetic rules cut off from the patients the supply of material by which the consumed portions of the body might be replaced, and that, consequently, the mortality is greater in Germany than in England, because the patients are starved, as it were. There is some truth in this assertion. I have no hesitation in saying, that the aggravation of a fever by giving the patient milk, eggs, and meat, has not been proved by actual observation, and I believe that much injury may be done by blind faith in the correctness of this hypothesis. There is no doubt that in every fever the consumption of the constituents of the body is greatly increased, and that no sort of exercise will use up the body so rapidly as a fever does. While continued bodily exertion is usually borne with impunity, because the increased consumption is hidden by increased supply, most fatal cases of fever are due to insufficient material being furnished for the replacement of that used up. Regarding typhoid fever, particularly, we find that in this disease the bodily temperature is above the normal for several weeks, and the consumption of tissue, by which this calorification is induced, is greatly increased. We see that in the most favorable cases, during convalescence, the greatly debilitated patients, who have often lost ten to twenty pounds in weight, recover very slowly; and we must agree, that these facts urge us to give meat, milk, eggs, etc., rather than water-soup, until it shall be proven that such diet increases the fever. But, on the other hand, it cannot apparently benefit the patient if we give him this nourishment, and he is not able to assimilate it; on the contrary, it would hurt him to fill his stomach with food that will not be digested, but will decompose and cause irritation of the gastric and intestinal mucous membrane. We have before seen that dyspepsia is a constant accompaniment of all fevers. If we do not attend to this fact in regulating the diet of

typhoid cases, but expect that their stomachs will supply plenty of gastric juice to digest large quantities of protein substances, we shall not increase their strength, but shall add a complication which will augment the danger. From this we have the rule, supported by experience, that at first we should give typhoid patients small quantities of milk and strong broth several times daily, whenever they desire it. The longer the disease continues, and the greater the exhaustion of the patient, the more untiringly we must attempt to supply nourishing food, but always in small quantities and in fluid form. In addition to these dietetic rules, we should have typhoid patients washed all over with pure cold water, or with a mixture of three parts of water to one of vinegar. This should be repeated several times daily, and should be done very carefully, so that its beneficial and soothing effects may not be annulled by the fatigue induced by the act itself. In a mild, regular course of typhoid, we need give no medicine; but probably the old prescription of dilute chlorine-water (aqua oxymuriat. ℥ ij, aqua destill. ℥ vj, det. ad vitr. nig., tablespoonful every two hours), or of muriatic acid in mucilage (acid. muriat. conc. ʒ ss, mucil. salep ℥ vj, syr. simp. ℥ j, tablespoonful every two hours) is at least of palliative service; and, as there are few patients that can be treated entirely without medicine, we should prescribe the above mixture, and it deserves the preference over other more active prescriptions. We may also follow the old custom of giving the muriatic acid in a weak infusion of ipecac. (gr. viij to ℥ vj), instead of in simple mucilage, during the second week, if the diarrhœa and bronchitic symptoms increase, and in a weak decoction of Peruvian bark during the third week.

The above treatment answers for most cases; but there are many others which require a different procedure, and in which threatening danger can only be averted by energetic treatment. In abdominal as in exanthematic typhus and other infectious diseases, the greatest danger is from the severity of the fever. But against this we have a most efficient remedy. The danger from the fever is a double one. On the one hand, the increase of bodily warmth above a certain point induces paralysis of the heart and renders life impossible; on the other hand, continued increase of the production of heat, or, what is the same thing, protracted increased transformation of tissue, on which the feverish over-heating depends, induces consumption of the body of the patient. In fevers of proportionately short duration, as in the acute exanthemata, exanthematic and abdominal typhus, the danger from *increase of the bodily temperature* is, of course, more to be feared than that accompanying the *increased production of heat.* But, in combatting the former, we should not lose sight of the latter, or else we may injure the patient instead of benefiting him. Without fear of being

misunderstood, I may give this warning before urgently advising the *abstraction of heat* in treating either exanthematic or abdominal typhus; because, so far as I know, the hydrotherapeutic treatment of typhoid fever was first introduced and carefully observed in my clinic. Until within a few years, when the bodily temperature had risen to a dangerous height, and there was occasion to lower it, I have had the patients wrapped in cold, wet sheets, and the proceeding repeated at intervals of ten to twenty minutes, until the desired end was attained. After satisfying myself that the patients were unnecessarily annoyed by the repeated transportation from one bed to another, which was unavoidable in this operation, I have used cold baths in its place, and they are much more convenient. I found that they had the same effect, and were better borne by the patient than was the repeated envelopment of the body in wet sheets. But I could not hide from myself that, immediately after the bath even, there was occasional exhaustion along with the retardation of the pulse, sinking of the temperature, and clearing of the intellect. This exhaustion usually passed off quickly, and the disease finally terminated in recovery; but, along with such cases, others occurred where the exhaustion continued longer after the baths, and where early death made me doubt if I had actually benefited the patient, or if, while removing one danger, I had not induced another. Perhaps this anxiety was overstrained, but, after the careful observations made at my clinic by *Liebermeister* and *Immermann* as to the amount of heat passing from the patient into the water, the possibility of this danger cannot be denied. It is true we reduce the temperature of the body by cold sheets and cold baths, but we at the same time increase the production of heat. If this were not so, the patient would be cooled much more. I think it would be proper to compare the action of an energetic abstraction of heat to that of excessive exercise; then it will be asked if it be advisable to subject an already exhausted patient to this action. The question, is it not possible, by hydrotherapeutic means, to reduce the temperature of the body without exhausting the patients by an excessive increase of the production of heat? which I consider a very important one, has been solved by *Obernier* and especially by *Ziemssen*. I consider the discovery, that a far less energetic and less sudden abstraction of heat than was accomplished by the methods formerly employed by myself and others will reduce the temperature of a typhoid patient two degrees or more, is a very important advance in the therapeusis of the disease I have not observed the above-mentioned state of exhaustion, which formerly alarmed me, since I have ceased to wrap patients in cold, wet sheets, or place them in cold baths, but have employed the following plan, recommended and tried by *Ziemssen:* As often as his tempera-

ture rises above 104°, the patient is placed in a bath, whose temperature is about 10° below that of his body, or about 94°. While the body and limbs are gently rubbed, we add cold water gradually till the temperature of the bath is reduced to about 68°. The patient is to remain about twenty or thirty minutes in the bath, till he is slightly chilled, and then to be placed quickly in a warm bed. At first, four or five baths daily are necessary, subsequently two or three. The method of *Ziemssen* has one great advantage, that in private practice there is no great objection made to it. The laity consider it much less objectionable to place a patient in a lukewarm bath than to wrap him up in wet sheets, or to pour cold water over him, etc. For moderating the fever in exanthematic as well as in abdominal typhus, next to the abstraction of heat, the administration of quinine deserves most confidence. This remedy has been repeatedly recommended in abdominal typhus, and almost as often it has been given up, because it did not equal the expectations of the employers. The only effect of quinine on the disease is to moderate the fever. If the temperature of a typhoid patient rise above 102°, I give quinine, but of late do not give such large doses as formerly, when I prescribed ten-grain doses and during the day gave as much as thirty grains. Now I usually prescribe one or two grains at a dose, in solution with dilute sulphuric acid. If we use quinine at the same time with the abstraction of heat, we are not obliged to repeat the latter so often, which is a decided advantage. *Wunderlich* has recommended digitalis as an antipyretic in abdominal typhus, and the results claimed by this trustworthy observer, in cases with frequent pulse and continued high temperature, urge us to further trials of this remedy, of whose antipyretic action, in the treatment of pneumonia and other inflammatory diseases, we have already spoken.

Next to the fever, the most dangerous symptoms, when extensive, are the disturbances of the respiratory organs, the bronchial catarrh, hypostasis, and collapse of the lung; but, unfortunately, we are far more powerless in regard to these dangers than in regard to that induced by the fever. The advice of most authors to give lukewarm, instead of cool drinks, when there is severe bronchitis, is based rather on theory than practice; when feeling the burning skin of a typhoid patient, no one would think seriously of combating the bronchitis by warm infusions. In severe typhous bronchitis, the most customary prescriptions are wet or dry cups, warm compresses, sinapisms, and blisters to the chest, and the internal administration of an infusion of ipecac. (gr. vij to ℥ vj), or of an infusion of senega (ʒ ss to ℥ vj), to which may subsequently be added liquor ammoniæ anisat. (ʒ ss — ʒ j). None of these prescriptions do much good, though the occasional ap-

plication of wet and dry cups most frequently causes temporary alleviation, and in most cases the cutaneous irritants are even injurious. If we hear extensive moist râles in the chest, while the expectoration is arrested, we may give a few doses of flores benzoës (gr. ij—iv), and, if they fail, give a certain emetic, which will be of service here, and, avert the threatening danger, if any remedy can. To prevent the extension of hypostasis, we may attempt to preserve the patient from constantly lying on the back, by changing him from one side to the other; but, unfortunately, this good advice cannot always be carried out continuously. The treatment of collapse of the lung and of pneumonia is the same as that of the bronchitis. In profuse epistaxis, we should not try mineral acids and cold applications too long, but apply a tampon early.

Among the intestinal symptoms, moderate diarrhœa requires no especial treatment. If the evacuations become very profuse, we may give astringents, especially solutions of alum (ʒ j to ℥ vj), or of tannin (℈ j — ʒ ss to ℥ vj), with an addition of tinctura opii (℈ j—ʒ ss). For the tenesmus, which is occasionally very annoying, starch injections containing ten to twelve drops of laudanum are almost always beneficial. Where there is much meteorism, we may attempt to evacuate the gas by passing a stomach-tube up the anus. Constipation is to be overcome by injections, or by castor-oil. In intestinal hæmorrhage, we should apply cold or ice compresses to the abdomen, and renew them frequently, at the same time giving alum internally in the form of serum lactis aluminatum. In perforation of the intestines, we should also employ cold compresses to the abdomen, but especially should give opium in quickly-repeated large doses (a grain every hour or two). At the same time, the patient may take small quantities of ice-water, or small portions of ice, to quench his thirst, but should have no food at all for several days. If peritonitis occur, independently of perforation of the bowel, cold compresses over the abdomen still deserve the preference to leeches. If there be paralysis of the detrusor vesicæ, which, to the great injury of the patient, is often overlooked by inexperienced and careless physicians, the bladder should be evacuated at least twice daily by the catheter.

It has already been stated that scrupulous cleanliness greatly aids in preventing bed-sores. On the first appearance of erythema, we should protect the reddened spot from further pressure by an air-cushion, and wash it several times daily with lead-water, dilute brandy, or with red wine. Eroded spots should be covered with lead or zinc salve, or with ungt. tannicum (ungt. contra decubitum Autenreithii), and lightly touched with nitrate of silver. If there be a deep loss of substance, with unhealthy base, it should be treated, according to the

rules of surgery, with cataplasms, stimulating salves, red precipitate, or nitrate of silver.

If, in spite of all our care, the strength of the patient sink more and more, if the pulse become small, the collapse and prostration dangerous, we should boldly give strong wines, such as madeira, port, etc., or strong beer (*Pfeufer*). No analeptic or roborant medicine in the pharmacopœia has an effect equal to that of a strong wine. The fear of increasing the fever by administering alcoholic liquors is ungrounded; and it is best not to wait for great exhaustion before giving wine, but, in all cases where the patients begin to grow weak toward the end of the second or commencement of the third week, to give them half a pint of light wine daily.

Lastly, during convalescence, the diet of the patient should be most carefully watched. The number who die during convalescence from typhoid fever, from the fault of the physician, because he has neglected to say exactly what and how much they may eat, or from their own, because they have not followed the rules laid down for them, is proportionately large. It is best to let the patient eat frequently, but only a little food at a time, so that the slight amount of gastric juice secreted by the convalescents may suffice for its complete digestion. All indigestible food, which forms large amounts of fæces, should be strictly forbidden. An apparently insignificant indigestion, a moderate diarrhœa, or slight vomiting, should be regarded as a very dangerous occurrence, because it may induce perforation of an ulcer that has not yet cicatrized.[3]

CHAPTER IX.

FEBRIS RECURRENS—RELAPSING FEVER.

Etiology.—Relapsing fever is among the acute infections about whose propagation by contagion there is no doubt; but we hesitate to proclaim this as a *purely contagious* disease, such as the acute exanthemata, measles, scarlatina, and small-pox, which never occur in a person who had not been infected by a measles, scarlatina, or small-pox patient. There are certain points in favor of the view that the infecting substance, which induces relapsing fever, is not only produced in the body of the patient, but that it may be produced outside of the human body. The mere fact, that certain regions, whose telluric conditions give rise to a pure miasm, malaria, are also subject to epidemics of relapsing fever, can hardly be made to agree with the ex-

tension of the disease by contagion alone; this is still more true of the fact taught by experience, that, in places where relapsing fever has not been seen for years, if a famine occur, and, from lack of proper food, people are obliged to eat bad or spoiled provisions, the disease will not unfrequently break out. On the other hand, there are grave objections to this double mode of origin and extension. The same reasons that satisfied us that the contagion in other infectious diseases consists in low organisms, are just as valid in relapsing fever; and, although we cannot say it is impossible, we are still very averse to supposing that organisms which reproduce themselves in the human body may also develop and increase outside of it under such different conditions.

The great resemblance in the mode of extension of relapsing fever and in its symptoms to the forms of typhus already treated of, cannot escape notice. On the other hand, there are some points indicating a difference between relapsing and typhus fevers, and a certain relationship to intermittent fevers. This circumstance, as well as the fact that there is no case known where a patient with typhoid or typhus fever has communicated relapsing fever to another person, and *vice versa*, has decided the best authorities to rank febris recurrens as a third form of typhus after abdominal [typhoid] and exanthematic [typhus] typhus fever. Now, if, after typhoid and typhus fevers had long reigned, relapsing fever occurred with them or in their place, this might probably be simply due to a modification of the low organisms forming the contagion of typhoid or typhus fever, induced by the vicinity of a marsh or the effect of bad food; in other words, to the development of a new species allied to but differing from the former ones.

I believe generally that the history of epidemics strongly supports the correctness of *Darwin's* theory of the origin of new species. For my part, I have no doubt that, in the course of centuries, new infectious diseases have developed and taken the place of others that formerly prevailed. In the writings of the ancients we find wonderfully-accurate descriptions, even of forms of disease, whose recognition and distinction offered the greatest difficulties. There can be no doubt that the regular and easily-recognized combination of symptoms characterizing measles, scarlatina, typhoid fever, etc., would not have escaped the sharp observation of *Hippocrates*, if it had existed then as it does now about the native place of the great Asklepiad.*

If the correctness of this hypothesis be accepted, and the depen-

* But in the writings of Hippocrates there is one place which seems to indicate that febris recurrens occurred even in his day.

dence of contagious diseases on an infection of the body from low organisms be regarded as certain, the appearance of new infectious diseases must seem a proof of the development of new species of organic beings. Should not the varied malignity shown by epidemics of the same disease at different times, and certain peculiarities of the individual epidemics, be most simply referred to *slight* modifications, and the gradual passage of certain phases of infectious disease in other forms as *extensive* modifications of the low organisms?

The susceptibility to relapsing fever seems very general, and it has often been noticed that almost every dweller in even large houses exposed to the contagion was attacked by the disease. No age escapes the affection; but most cases are among the young and middle-aged. The predisposing influence of bad dwellings and insufficient food is not to be mistaken. Poor people furnish so large a contingent of the cases of relapsing fever that this cannot be explained solely by their numerical preponderance over the well-to-do classes. People in good circumstances have no immunity from this disease, and nurses and physicians attending patients with it are not unfrequently infected. One occurrence of relapsing fever does not seem to remove the liability to it, as it does in other forms of typhus.

Anatomical Appearances.—The result of autopsies made in epidemics of febris recurrens of different intensity and malignity, together with symptoms observed during life, justifies the following conclusions: The infection of the organism by contagion not only induces high fever running a peculiar course, but also pathological changes in various organs, especially in the spleen, liver, and kidneys. Usually these pathological changes consist only of disturbances of the circulation and in such changes of structure as may be readily removed. But in malignant epidemics more extensive structural changes develop in the above organs, bearing partly the character of parenchymatous, partly of suppurative inflammation. The disturbances of nutrition due to relapsing fever have not a specific character, and do not differ from those induced by other injurious influences.

The corpses long retain a rigor mortis which occasionally begins very early. The skin is usually light yellow, sometimes typically jaundiced. At the dependent parts of the body there is extensive *post-mortem* hypostatic congestion.

The muscles are not so dark as in other forms of typhus. On microscopical examination a more or less extensive degeneration of the muscular fibrillæ will be found; they appear cloudy, indistinctly transversely striated, filled with molecular fine granules, which disappear on the addition of acetic acid, while the filaments become pale, and show neither transverse nor longitudinal striæ (*Küttner*). This degen

eration is not confined to, or even chiefly located in muscles, which, during life, were the seat of severe pain, but, when found at all, usually extends to all the muscles of the body.

In recent cases the blood is dark cherry-red, and contains but few fibrinous clots. In protracted cases the blood is watery and without a trace of coagulation.

When death occurs at the height of the disease, the brain and meninges are vascular and dry, the ventricles empty. If death does not result till the later stages of the disease, the meninges are slightly injected, the brain itself bloodless and pale, the subarachnoid spaces and lateral ventricles containing quantities of serous fluid.

Some observers (*Kremiansky*) found hæmorrhagic pachymeningitis very often, partly in its commencing stage, partly as an extensive hæmatoma of the dura mater.

The bronchial mucous membrane presents no constant changes; the lungs are more or less congested, especially at the dependent parts; in protracted cases they sometimes contain hæmorrhagic infarctions or extensive pneumonic infiltrations.

The heart is flabby, pale, and friable. On microscopical examination, *Küttner* found it infiltrated with an albuminous or fibrous mass. If the disease has lasted long, the walls of the heart are occasionally much thinned.

As a rule, the gastric and intestinal mucous membrane is reddened by injection and ecchymosis. The intestinal glands are sometimes enlarged, but never sloughing. The ductus choledochus is occasionally closed, by swelling of its mucous membrane, and obstructing masses of mucus. In such cases the contents of the intestine are but little colored, while the gall-bladder is distended.

The liver is considerably enlarged, apparently from the increased amount of blood in it. According to *Küttner*, it sometimes has a marbled appearance, distinct, yellowish-white, wax-like spots appearing in the normal parenchyma; when far advanced, these resemble nodules of medullary cancer. At such points the individual acini are not recognizable. The liver-cells have lost their polygonal form; the nuclei are perceived with difficulty, and filled with homogeneous contents. In cases beginning with icterus, and proving fatal in the first attack (bilious typhoid), the same author found the liver smaller, the parenchyma jaundiced, much relaxed, and broken down. There were the same microscopical appearances as in acute yellow atrophy of the liver.

The spleen also is enlarged; it may attain five or six times its normal size and weigh four pounds or more; its parenchyma is only exceptionally as soft and fluid as in the first stages of the other forms

of typhus; usually it is hard and friable, and on section the Malpighian bodies project as grayish-white or jaundiced points the size of a pin's head. Occasionally in the spleen we find yellowish-white foci, roundish or irregular in shape, from the size of a hemp-seed to that of a hazel-nut, at first quite firm, but later breaking down into pus. *Küttner* considers these as ruptures in the cavernous venous meshes of the spleen, which subsequently, as in phlebitis after venous thrombosis, induce inflammation, suppuration, and putrefaction of the parts around.

The kidneys also are enlarged, sometimes to double their normal size; this is due solely to swelling of the cortical structure. It is difficult to detach the capsule, and, when this is done forcibly, small portions of the renal tissue remain adherent to it. On microscopical examination we find the epithelium of the uriniferous tubules swollen and filled with fine molecules; in the later stages the epithelial cells have been destroyed, and the tubules are filled with fine granular detritus. Rarely there are numerous small abscesses scattered through the kidneys.

Symptoms and Course.—The length of the period of incubation is not yet accurately determined. Many patients feel perfectly well from the inception of contagion till the outbreak of the disease, while others complain, a few days before the actual commencement of the malady, of ill-defined and uncharacteristic disturbances of their general health, of great weariness, pain and heaviness in the head, increased thirst, and occasionally of migratory pain in the extremities.

Whether the above prodromata have occurred or not, the disease itself almost unexceptionally begins with a chill of variable duration and intensity, followed by a persistent feeling of great heat.

With the commencement of the fever the patients become very weak, complain of pain in the head, especially in the forehead, often also of dizziness and tinnitus, and of severe muscular pains almost characteristic of the disease, which affect chiefly the neck and limbs, and are among the most annoying symptoms of the disease. A slight amount of psychical excitement, usually present at first, generally passes off. The feeling of illness is unmistakable, the patients showing no interest in their surroundings. Delirium and deep sopor are exceptional, even where the fever is very high, which is a strong argument against the exclusive dependence of psychical disturbances, in acute infectious diseases, on the height of the bodily temperature. At first, the face is slightly flushed, the skin hot and dry, rarely is it covered with slight perspiration. We must beware of regarding this moisture of the skin at the height of the disease as a favorable sign

(I have already stated that, of two patients with equal bodily temperature, the one whose skin is moist is the more feverish.) There is great thirst, but no appetite, although the patients do not obstinately refuse food. The tongue is broad, rounded anteriorly, and bears the imprint of the teeth along its sides; it has a thick, white coating, the edges and point are red. Even where the fever is high the tongue usually remains moist, and is scarcely ever covered by the dry, black coating visible on the small pointed tongue, and on the teeth, and gums, in typhoid fever.

The pharynx also is affected by catarrh, usually mild, but sometimes very intense, characterized by more or less injection of the mucous membrane and secretion of a tough mucus. In some cases bilious vomiting occurs at the commencement. As a rule, there is constipation; far more rarely, diarrhœa, perhaps from excessive drinking. The respirations are increased in proportion to the fever, and, without any complication of the lungs or bronchial tubes, may attain to thirty or forty, or even more, in a minute. There may be symptoms of bronchial catarrh or not. The abdomen is neither distended nor depressed; the regions of the liver and spleen are sensitive to pressure, and sometimes spontaneously painful. Physical examination early shows considerable enlargement of the liver and spleen. Not unfrequently the former extends almost to the navel and far into the left hypochondrium. The latter is also perceptible to the touch when it extends beyond the angles of the ribs.

According to the careful researches of *Obermejer*, in most cases the urine shows the peculiarities of parenchymatous nephritis. Besides more or less albumen, blood-corpuscles and dark epithelial cylinders were found in about two-thirds of his patients; later in the course of the disease the cylinders were covered with granular epithelial detritus, and finally perfectly pale cylinders were passed. As a rule, during the fever the amount of urine passed is diminished; its specific gravity is 1012 to 1020.

A slight degree of icterus, sometimes accompanying the above symptoms and depending on catarrh of the ductus choledochus, is readily recognized to be hepatogenous by the diminished color of the fæces and the discovery of bile-acids in the urine, and is to be distinguished from the icterus hereafter described, which is probably hæmatogenous and renders the disease malignant.

From the above description it is evident that, apart from the severe muscular pains, the patient's symptoms resemble those accompanying intense fever; and in fact we may regard the fever as the most prominent and characteristic symptom of this disease. This fever shows not only the peculiarities to which the disease owes its name, but it

has others which enable a certain diagnosis, even in the first attack.

From the observations of *Obermejer*, who has taken accurate measurements of temperature during the chill, with which the later attacks also usually begin, we may assume that the bodily temperature is elevated even during the initial chill, and that this is even preceded by a slight elevation of temperature. The bodily temperature attained is very unusual; it is not at all rare for it to reach 107.3°, and in some cases 109°, or an elevation that never occurs in other diseases, and at which many regard the continuance of life as impossible. This, like typhoid fever, has a remittent type. The daily variation is usually about one degree, the greatest differences being in the morning and evening. The pulse also is more frequent in relapsing fever than in almost any other disease. In nearly every case it rises to 110 or 120, not unfrequently to 120 or 130 and even to 150 beats per minute, without being in itself of very evil import. The frequency of the pulse corresponds far less than in other diseases to the bodily temperature. As a rule, the pulse is at first hard and tense, later, from paresis of the muscular coats of the vessels, it becomes soft, undulating, and not unfrequently dicrotic. With slight modifications the disease goes on thus for from five to seven, rarely longer than eleven or twelve days; then there is a sudden change. After a transient increase of all the symptoms, in which the bodily temperature reaches its highest point, the peripheral arteries pulsate strongly, and abundant epistaxis often occurs; the previously-dry skin breaks into a copious perspiration, and there is an alleviation of all the symptoms. According to *Obermejer*, the duration of defervescence is usually eight or nine hours, during which time the body cools off about 9°, so that, if before the crisis the temperature was 106.4°, afterward it will be 96.8°, or less than normal. I have no doubt that the excessive loss of warmth, caused by the evaporation of the perspiration, contributes materially to this rapid fall of temperature. During a critical sweat, the active flow of blood from the interior of the body to the surface will almost equalize over the body the loss of warmth that is induced by evaporation of the perspiration. Such a sweating *must* induce cooling, such as could not be attained in the same length of time by any hydropathic procedure. It does not lessen the loss of heat by conduction and radiation; and to this is added the great loss due to evaporation of the perspiration. I would call attention to the immense amount of heat withdrawn by evaporation of the abundant perspiration during a Turkish bath; for there, in spite of the surrounding air being 40° or more warmer than the body, and the continued heating from within, there is no considerable elevation of its

temperature. I should like to know if such a rapid defervescence of the fever is seen in cases where, instead of sweating, there is critical diarrhœa.

In the days following the crisis, the bodily temperature, which had almost always fallen below the normal, rises one to two degrees. The pulse varies from 48 to 50 beats a minute, the appetite returns, the tongue cleans off, the muscular pains subside, the patients feel stronger, and many of them wish to leave their beds.

But only in rare cases, where the disease is incorrectly termed relapsing, does this very agreeable condition (which, to the uninitiated, must seem a commencing convalescence) actually form the beginning of recovery. In most cases, in from six to eight days, rarely less, or from twelve to fourteen, there is a new attack, closely resembling the first. This second attack also usually begins with a chill of variable duration and intensity; the subsequent feeling of heat, debility, depression, and thirst, attains about the same degree as in the first stage of the disease. The muscular pains also recur, but are less severe; the tongue, which had cleared off, is again coated, the appetite is lessened. The spleen and liver, which during the interval had somewhat decreased in size, again swell, and occasionally become even larger than in the first attack. During this second paroxysm, the bodily temperature rises just as high as, in many cases higher than, in the first, but the pulse is usually less frequent.

The second attack is commonly shorter than the first, lasting only three or four days. The crisis terminating it has about the same symptoms as that closing the first attack. The disease generally ends with the second attack. Far more rarely it is followed by a third, fourth, or even fifth, which have essentially the same symptoms.

By far the most common termination is in recovery; the very slight mortality, which in most epidemics is only two or three per cent., and rarely higher than six or eight per cent., is in great contrast to the severity of the symptoms, especially to the temperature. If the disease prove fatal, death results either during the attack, from collapse and general paralysis, or more rarely during the interval, from exhaustion, or lastly as a result of secondary diseases and complications, among which pneumonia is the most important.

Pastau, as well as *Obermejer*, has given us valuable investigations as to the weight of the body in relapsing fever. These show that during the disease the body loses from a tenth to a fifth of its weight, and that this cannot be solely referred to the diminished supply of nutriment. Exact investigations prove again the correctness of the doctrine that increased transformation of the constituents of the body plays the most important part in the elevation of temperature during

fever. It is very interesting to me to find that *Obermejer* has proved that there is a steady decrease of the weight of the body during the attack, but that it is far greater and more rapid on the critical days. These observations agree perfectly with those which were made in my clinic by Dr. *Steiner* even before the *Leyden* investigations, and which led to the following conclusions: Where the bodily temperature is only moderately increased, the loss of weight may be less than that observed during the same length of time in healthy persons. This is because during fever the function of the perspiratory glands, as well as of other secretory organs, is suspended, and the loss of bodily weight from evaporation depends chiefly on evaporation of the perspiration. But, if the bodily temperature rise to 102° or 104°, the loss of weight is always greater than in healthy persons under circumstances as nearly as possible the same. When the temperature of the body is so high, even if the secretion from the sweat-glands be entirely arrested, more fluid is evaporated from the mucous membrane of the respiratory passages than is usually evaporated from the surface of the body and the air-passages together of persons with normally-acting sweat-glands. The most rapid decrease of bodily weight occurs at the defervescence of the fever, for then the sweat-glands again act, while the still-elevated temperature is accompanied by accelerated evaporation. I believe in the correctness of the idea that, at the height of the fever, there is only a relative retention of water in the body, but do not think it necessary to refer this retention to diminished action of the sweat-glands.

Under certain influences still unknown, possibly merely as a result of the action of a particularly intense contagion, relapsing fever assumes a very malignant character. The appearance of the disease is especially modified by excessive participation of the biliary apparatus, and in most cases death appears with severe symptoms. *Griesinger* describes this malignant form of relapsing fever, from his observations made in the East, and terms it bilious typhoid. The St. Petersburg epidemic of 1864 to 1866, where, besides simple recurrent fever, there were numerous cases of bilious typhoid, especially at its commencement, fully confirmed *Griesinger's* description of the disease, as well as his opinion that it was a severe form of recurrent fever.

At first the symptoms vary little from those of simple recurrent fever, except that there are more depression and headache, and greater mental sluggishness, bilious vomiting is more frequent, and the tongue shows a tendency to dryness, while in simple recurrent fever it is apt to remain moist during the whole course of the disease. Usually, after a few days there is diarrhœa, with evacuation of bilious looking masses; or else the passages have a dysenteric appearance

Often also there is bronchial catarrh of variable intensity; and generally from the fourth to the sixth day of the disease, after the liver and spleen have swollen considerably, and become painful, there is severe icterus without discoloration of the fæces. About this time the prostration of the patients is very great; they are perfectly apathetic and somnolent or delirious. The tongue is dry and crusted, pulse slow, the hot skin loses its turgescence; and, in this state, many die with the symptoms of sudden collapse.

In others, at about the same time as in simple recurrent fever, there is a more or less complete crisis, followed by a rapid improvement of all the symptoms. The patients seem to be convalescing, til. a relapse occurs with the former symptoms, and they generally soon succumb.

Finally, in other cases there is no crisis, and in the second week, except for the intense icterus, we are reminded of severe and protracted cases of other forms of typhus. According to *Griesinger's* description, this period of the disease is characterized by more or less profound sopor, delirium, great sensitiveness of the abdomen, involuntary passages of dark, thin fæces, or markedly dysenteric dejections, or occasional large evacuations of clotted blood, difficulty of swallowing, croupous coating on the pharynx, bronchitis, extensive lobular pneumonia, occasional pericarditis, petechia and miliaria of the skin, and irregular chills. It almost always terminates with slight convulsions, sometimes by sudden collapse, internal hæmorrhages (rupture of the spleen), or secondary disease of the thoracic organs. Rarely, the patient recovers either with a rapid change, or slowly and irregularly —the latter is especially the case if there has been extensive disease of the thoracic or abdominal organs, such as pneumonic infiltration or dysentery.

Treatment.—The prophylactic rules for preventing the spread of or protecting one's self against relapsing fever appear at once from what we have said of its etiology. Improvement of the condition of poor people, care for healthy nourishment, and roomy dwellings, necessary as they are for prophylaxis, will probably never be fully carried out, as it is so much easier to separate the healthy from the sick, as is required by the contagiousness of the malady.

The slight mortality contraindicates an energetic treatment of the disease. Thus far attempts to cut short the process and prevent relapses by quinine have failed. In the same way digitalis did no especial good in the Berlin epidemic.

By cold baths, in recurrent fever also, we may almost always lower the temperature from one to five degrees; but this usually continues only a short time, the bodily temperature soon rising again to its

previous height or even higher (*Obermejer*). I have repeatedly said that I consider energetic abstraction of heat by cold baths quite a heroic remedy. If the high temperature in recurrens were accompanied by great danger to life, of course we should have no hesitation in combating the exhaustion of the body, from the increased production of heat, by the energetic and continued use of cold baths; but, since, in spite of the high temperature, relapsing fever has a low mortality, scacely three per cent. of the patients dying, I do not consider cold baths indicated in this disease. It will be well to limit ourselves to sponging the body with cold lotions, and, if the cerebral symptoms be severe, to the application of ice to the head. At the same time we should attend most carefully to the patient's cleanliness and supply of fresh air, and internally give dilute mineral acids. *Obermejer* recommends the administration of lemon-juice when the kidneys are much affected. We should carefully give the patients nourishment early, and, if there be much debility, prescribe some wine. From the tendency to diarrhœa, they should not drink much water. During convalescence, iron and quinine may be given.

Griesinger recommends large doses of quinine (gr. x–xxx daily) in bilious typhoid. He says it is just as useful here as in intermittent fever; but it is well not to begin the quinine treatment till after the administration of a mild purgative of salts, oil, or cream of tartar, at the commencement of the disease.

CHAPTER X.

EPIDEMIC DIPHTHERITIS—MALIGNANT PHARYNGITIS—ANGINA MALIGNA—DIPHTHERIA.

Etiology.—Epidemic diphtheritis belongs among the infectious diseases, and even among those that are most typically *contagious*. The *miasmatic* origin of the disease is doubtful, at least in our country, where it has only occurred during the past ten years, and has appeared almost exclusively as more or less extensive epidemics which occasionally spread around from one place to another. The contagion is contained in the false membrane and shreds of tissue detached from the fauces, and in the air breathed out by the patient. Physicians are in great danger of being infected by the morbid products coughed out by the patient when they are painting or cauterizing his throat, or performing a tracheotomy. Science has to mourn the loss of a series of excellent physicians and observers who fell victims to diphtheria while

in the line of their duty. The numerous cases of infection of persons who have been in the same room with diphtheritic patients, without coming in their immediate vicinity, prove that the air exhaled by the patient, which does not contain shreds of exudation or tissue, is a vehicle for the contagion. Further observation must decide, if, besides the above vehicles for the poison, there be still others; how tenacious the infection is; whether persons who do not themselves take the disease may carry it to third parties, etc. The predisposition to diphtheria is unmistakably very extensive. The greater frequency of the disease among children than adults does not seem to me due to greater predisposition of the former, but to their being more exposed to infection than adults are.

Anatomical Appearances.—Just as in other infectious diseases, in diphtheritis also certain organs are chiefly subjected to the disturbances of nutrition, and (to employ our former mode of expression) diphtheritis is constantly localized in the fauces, less constantly in the upper portion of the air-passages, in the kidneys, spleen, and in some very obscure manner in the nervous system. The disturbances of nutrition in the above organs do not anatomically correspond. In the pharynx we find the form of diphtheritic inflammation to which the disease owes its name, and which we have often described. The fauces, and especially the tonsils and soft palate, are covered with grayish-white pseudomembrane, which is not easily removed, and which leaves an ulcerated loss of substance, when it finally breaks down into a discolored fetid mass, and falls off. The diphtheritic pseudomembranes, or, to speak more precisely, the diphtheritic sloughs, result from superficial gangrene of the mucous membrane, which again depends on compression of its nutrient vessels by an interstitial fibrinous exudation, or from swelling of the tissue-elements, which are filled with a cloudy substance. If the larynx and trachea participate in the disease, the croupous, not the diphtheritic, form of inflammation of the mucous membrane occurs; that is, the surface of the mucous membrane is covered with a more or less tough and consistent false membrane, which may readily be removed, and leaves no loss of substance after its removal. This circumstance has induced some physicians to identify primary genuine croup, which is due to catching cold, etc., with croupous laryngitis caused by infection with diphtheritic contagion. I consider this a false view. The division of diseases, according to the pathologico-anatomical changes they induce, is only a make-shift. In all cases where, as in genuine and diphtheritic croup, we find that two anatomically similar disturbances of nutrition depend on very different causes, we should consider them as distinct. A small-pox pustule may very much resemble one induced by rubbing antimonial ointment

into the skin; a pemphigus bleb may resemble a blister induced by a circumscribed burn, but no one would assert that these scarcely-distinguishable disturbances of nutrition were due to the same disease. The previously-described parenchymatous degeneration of the kidneys is found in about half of the cases. The spleen is usually enlarged and soft. The anatomical changes of the nerve centres, or peripheral nerves, on which diphtheritic paralyses depend, have not yet been discovered. We know as little about the anomalies of the blood in diphtheritis as in other infectious diseases.

Symptoms and Course.—The disease almost always begins with apparently insignificant and harmless symptoms. In some cases the general health is disturbed a few days before the disease breaks out; the appetite is less; the patients complain of dulness, depression, and chilliness. More rarely the disease begins with a severe chill, accompanied by nausea and vomiting. At the same time the patient complains of difficulty of swallowing; but in most cases this is no greater than it usually is, in simple catarrhal angina. If the fauces be not yet covered with false membrane, but only somewhat reddened and swollen, at this stage we can only suspect or recognize the disease, when we know that diphtheritis is epidemic, or that persons about the patient have or have had the disease. A very suspicious, and usually a very early, symptom is a hard swelling of the lymphatic glands lying at the bifurcation of the carotid artery, which, as *Luschka* especially insists on, are directly connected with the lymphatic vessels of the soft palate. Not unfrequently the persons about the patient have their attention first called to the disease by swelling of these glands, the complaints of slight difficulty of swallowing having passed unnoticed. It is far more common for us to find more or less extensive grayish-white patches in the fauces at our first examination, than to have the opportunity of examining the throat at the commencement of diphtheritis, before the formation of false membranes has begun. And then we can have no doubt about the dangerous and malicious foe with which we have to deal. Even in cases where the disease has begun without a chill, where the fever is slight, or entirely absent, where the general health is excellent, so that the patient can hardly be kept in bed, even where the difficulty of swallowing is very insignificant, and where the deposits are very slight, are thrown off without putrescence, and leave a scarcely-perceptible loss of substance, we are not at all sure that the disease will run a favorable course, that the dangerous accidents, of which we shall hereafter speak, will not occur, and that there will be no paralysis during convalescence. The fact, which I have frequently observed, that albuminuria occurs during the above cases, which run their course without fever, shows that the parenchymatous

degeneration of the kidneys in diphtheritis is not due to excessive increase of the bodily temperature, but is a direct result of the infection with diphtheritic poison.

If the disease has begun violently, if a chill and repeated vomiting have been the first symptoms, the subsequent course of the disease also is usually more severe. It is true, the difficulty of swallowing generally remains moderate, and the fever does not become very high, but the patient grows pale, the eyes become dull, the pulse small, and usually frequent, rarely retarded; the patients are very sluggish and apathetic. In many cases the putrefaction of the false membrane causes a penetrating, foul breath. If the nasal mucous membrane participate in the disease, a discolored, fetid fluid flows from the reddened and eroded nostrils. The swelling of the cervical glands becomes more marked; the enlarged glands are very hard and resistant, but have no tendency to suppurate. In about half the cases, examination of the urine shows the presence of a considerable amount of albumen. Even, after a few days, death may occur from general paralysis, while the intellect remains clear. Some patients, whose state had not excited any anxiety, and whose general condition was previously very satisfactory even, die unexpectedly with the symptoms of general collapse, without our being able to find any explanation for the occurrence. Sometimes also attacks of deep syncope occur, and pass over, till finally a new attack ends in death. Even the last-mentioned severe cases may terminate favorably. Then the false membranes are detached, and no new ones are formed; the remaining ulcers clean up and cicatrize. At the same time the difficulty of swallowing disappears, the glandular enlargement subsides, the depression of the patient disappears, and, if there be no sequelæ, perfect convalescence follows in two or three weeks; but it is usually a long time before the patients recover entirely.

The disease, as above described, is greatly modified when the diphtheritic inflammation of the fauces is accompanied by croupous inflammation of the larynx and trachea; for then the above symptoms are complicated with hoarseness, aphonia, excessive dyspnœa, and other symptoms described when speaking of genuine croupous laryngitis. This complication occurs in the mild, as well as in the severe cases. Frequently, examination of the fauces and the epidemic occurrence of diphtheria alone enable us to decide to which form of croupous laryngitis the case belongs. Even such cases may end in recovery; but most of the patients die, some with the symptoms of collapse, some with those of insufficient respiration, and poisoning of the blood with carbonic acid.

Even when the disease apparently ends in recovery, it is often fol

lowed by paralysis. The not unfrequent occurrence of diphtheritic paralyses after very mild cases, as well as the curious fact that they never follow the disease immediately, but come on from two to four weeks after its disappearance, sufficiently explains why the connection between the paralyses and the diphtheritis so long escaped recognition. Paralysis of the soft palate and pharynx is the most frequent form of diphtheritic paralysis, and generally precedes the other forms; when the soft palate is paralyzed, the patients have a nasal voice; on attempting to swallow fluids, they enter the nose. If the pharynx also be palsied, the act of swallowing is greatly impaired, and we are sometimes obliged to nourish the patient through a stomach-tube. This paralysis of the muscles near the seat of diphtheritic inflammation is most frequently accompanied by that of the muscles of the eye, by which the power of accommodation is lost, and the patients begin to squint. The extremities also, especially the feet, are occasionally attacked by more or less complete paralysis. In a very small epidemic I saw two cases of total paralysis of all the extremities. The prognosis of diphtheritic paralysis is generally favorable; almost all cases recover sooner or later. The various attempted explanations of these cases are unsatisfactory. We do not even certainly know whether they are of peripheral or central origin. It has been suggested that the paralyses after diphtheria are analogous to those occasionally observed after other severe diseases, especially after severe typhus. But this suggestion is opposed by the great frequency of diphtheritic paralysis compared to that after other diseases, as well as by the marked disproportion between the intensity of the malady and the consequent paralyses, distinguishing diphtheritic paralyses from those remaining after other diseases.

Treatment.—Prophylaxis requires that the physician should protect himself from contact with the false membrane and shreds of tissue that are coughed up, and that he should warn the attendants on the patient of the danger of this contact. When circumstances permit, those who have nothing to do with the care of the patient should keep out of the sick-room.

The recommendations of the varied internal and external remedies that are said to have proved efficacious against diphtheritis, have usually originated in the last stages of epidemics, at which time the cases are usually milder, and recoveries more frequent, even without treatment. Almost all physicians, experienced in the treatment of diphtheria, agree that, in severe attacks, the most prized remedies are perfectly useless. In recent cases I deem it advisable to remove the false membrane carefully, and touch the dried bases with nitrate of silver, concentrated muriatic acid, or liquor ferri sesquichlorat., but not to re-

peat this operation more than once or twice daily, and not to continue it too long. Besides this, as long as the mucous membrane is still very red and swollen, I let the patient swallow small pieces of ice slowly, and prescribe a solution of chlorate of potash (~~℥ i to ℥ vi~~), with directions to take a tablespoonful in the mouth every two hours, and to keep it in contact with the pharyngeal tissues for some time before swallowing it. I have no personal experience about the local application of chlorine-water, or the solutions of sulphite of soda (℥ i to the ℥ j), or of permanganate of potash, which are said not only to remove the fetid odor, but also to arrest the putrefaction. On account of the threatening collapse, we must avoid all debilitating treatment, especially abstraction of blood; on the contrary, we should, from the first, attend to keeping up the strength of the patient by tonics, quinine, and iron, and by wine and nutritious diet. If collapse occur, we should give analeptics, camphor, musk, and particularly plenty of strong wine. Croupous laryngitis coming on during the disease requires about the same treatment that we advised in the first volume for idiopathic croupous laryngitis; only I do not employ abstraction of blood or calomel even in recent cases. Tracheotomy should not be deferred too long, if we hope to have it succeed, which it rarely does.

Short, cold ablutions, cold douches, and sea-bathing, have obtained the most confidence in diphtheritic paralysis. The induced and constant currents have also been advised. I have subjected many cases to long-continued treatment by electricity without benefit. In one case, where the patient was paralyzed in all her extremities, and was treated at my clinic by electricity, without benefit, for four weeks, she completely recovered, without any treatment, a short time after being dismissed from the hospital.[4]

[Diphtheria so often occurs without our being able to detect the source of contagion, that we may very strongly suspect its occasional spontaneous origin.

Some authors regard general blood-infection as the first step, and say the throat-affection is only a local expression of this, as the bronchitis is of measles or pharyngitis of scarlatina; while others say the affection is at first local, and may remain so or may become a general disease. Numerous vegetable organisms (micrococci) have been found in the diphtheritic membranes and even in the blood of the patients. The transmissibility of diphtheria to the lower animals by inoculating in the muscles, trachea, cornea, etc., has been proved, and the excessive multiplication of bacteria at these points has indicated them as the bearers of the contagion. In favor of the first view is the fact that at the commencement of

the disease there may be high fever, but no change perceptible at the point where the diphtheritis subsequently appears; also that in scarlatina, which is a primary blood-disease, diphtheria often occurs. The claims for the local origin of the disease are supported by the results of inoculation, which tend to show that the disease starts from the point of inoculation and spreads through the body. It has been asserted that these experiments prove nothing certainly, and that inoculation of other decomposing substances may induce the same changes as the diphtheritic membrane causes. If the diphtheritic poison entered the lungs and was thence transferred to the blood in some cases, and located and was propagated in the pharynx in others, it would perhaps explain both classes of cases. Some English observers consider it as established that milk is frequently an infectious agent; whether the poison is generated in the cow itself, or whether the milk is merely a carrier of germs taken in the cow's food, is not decided.

Persons in the vicinity of a diphtheria patient may have a catarrhal angina due to the poison. A second form is croupous diphtheritis, with a membrane which may be readily detached from the subjacent mucous membrane. The third and severest form is where the fibrinous deposit is both on and in the membrane; between the fibres are pus- and blood-cells, micrococci, etc.; the mucous membrane is quickly destroyed, leaving gray or brown sloughs, which render the breath very foul; this slough may extend to the subjacent cartilage or bone.

Deeper in the air-passages, diphtheria is croupous in nature; the ulceration of the mucous membrane does not usually extend below the larynx, that is, where pavement changes to cylindrical epithelium. In persons who have died of diphtheria, the blood is generally imperfectly coagulated, dark, and tar-like. Changes in the kidneys are very common.

The latent stage of diphtheria is usually short, though cases occur where it is possibly of some weeks' duration. The catarrhal form has the same symptoms as ordinary angina, and it may be only the surrounding circumstances that would lead us to suspect diphtheria; it may run its course in this way, or become a severe disease. *Croupous* diphtheria is usually accompanied by high fever; there may be burning in the throat or pain on swallowing; if these symptoms be absent, the disease may for some time escape recognition. Inspection shows the mucous membrane to be dark red and swollen, with deposits of false membrane, which develop rapidly, and may even cover the whole pharynx and extend into the nares or middle ear, but especially into the larynx, or else may remain confined to the pharynx. The latter cases are apt to terminate

favorably, the false membranes being detached and the fever subsiding in a few days. Such cases are common in youths and adults, but advance of the disease to the larynx is more to be dreaded in infants. As the first deposits clear off, others may take their place.

In septic or putrid diphtheria there is infiltration and mortification of the mucous membrane itself, with consequent ulcers and gangrenous loss of tissue. The detached fragments putrefy and give a foul odor to the breath; the submaxillary and lymphatic glands are swollen; there is fever characteristic of septicæmia; and usually the patient dies in a few days. But a line of demarcation may form around the necrosed parts, and recovery take place. If the disease attacks the nose, it causes a running, which may be mistaken for cold in the head till the discharge becomes bloody or sanious.

Diphtheritic paralysis may not occur till four or five weeks after the disease; it is peripheral in character, as is shown by the diminution of electro-muscular excitability; it lasts several weeks or even months; it most frequently affects the muscles of the pharynx and eye; the prognosis is usually favorable.

In the treatment, various disinfectants, such as chlorine one part to six of water, weak solutions of carbolic acid, salicylic acid, permanganate of potash, also alcohol, etc., may be employed locally in conjunction with hot vapors, which hasten suppuration, and possibly the pus may wash off the micrococci; these hot vapors may be tried every half hour or so. We should avoid rough or frequent cauterizations, for fear of increasing the inflammation. In diphtheria of the larynx we may use sprays of lime-water and salicylic acid. Tracheotomy has rarely proved a successful operation in this disease. Internal administration of chlorate of potash and tincture of muriate of iron, properly diluted, and in doses suited to the age, every half hour to two hours, is perhaps one of the most favorite methods of treatment.]

MALARIAL FEVERS.

CHAPTER XI.

INTERMITTENT FEVER.

Etiology.—Intermittent fever results from infection of the body with a poisonous substance called marsh miasm, or malaria. Malaria is not a product of decomposition (see below), but the decomposition of *vegetable* substance has an unmistakable influence on the development or excessive increase of this poison. I shall only adduce the following circumstances in support of this fact: Malarial fevers are

endemic chiefly in marshy regions, and in such places the number of persons affected increases or decreases according as circumstances favor or do not favor the decomposition of dead vegetable matter in the marshes. If it grow very cold, so that the marshes freeze up, the intermittent fever ceases. The same thing occurs when, in dry seasons, the marshes entirely dry up, or if, in very wet seasons, a thick layer of water protects the mouldering bottom of the marsh from the action of the sun and air. On the other hand, in marshy regions, hot seasons, if not too dry, so that the sun's rays can act freely on the exposed but still moist bottom of the marsh, are characterized by the great prevalence of intermittent fever. It is uncertain whether the decomposition of certain vegetables, or a peculiar quality of the water, favors the development of malaria more than other causes. A mixture of sea-water with spring and rain water—which occurs in marshes near the ocean, from high tides or heavy winds forcing the sea-water into the marshes—so-called brackish water, appears to be peculiarly injurious, because many fresh-water plants, as well as many marine plants, contained in the mixture, cannot thrive in it, and consequently die and decompose. Malarial fever is endemic in low lands near rivers, which are flooded yearly, just as in marshy regions. No especial explanation is necessary to show that this flooding also causes the death of quantities of vegetable matter, which subsequently decomposes when exposed to great heat. Intermittent fever occurs where land has lain for a long time uncultivated, and is then broken and tilled again, from the same causes as it does in marshy regions; for here, too, quantities of dead vegetable matter are brought up and undergo decomposition. Lastly, in some cases where intermittent fever occurs under apparently opposite circumstances, that is, when it is very dry, it has been shown that the soil was rich in subsoil water, and that, under a dry, porous surface, parched by the summer heat, there were subterraneous swamps. These common peculiarities, which appear in most of the regions where malarial fever prevails, do not, however, as was before said, justify us in concluding that malaria is a chemical body, an organic or inorganic, a solid or gaseous product, formed by the decomposition of vegetable substances. On the contrary, the non-occurrence of intermittent fever, in some typically swampy places, and its appearance in some places where extensive or specific putrefaction would be most unlikely to take place, seem to indicate that while the conditions peculiar to swamps, marshes, etc., favor the development of malaria, they are not a *sine qua non*, nor do they alone suffice to induce the disease. This view is even more strikingly supported by the observations that all the persons drinking water from a certain swamp were taken sick with intermittent fever, as these observations contrast with a large number

of others, where the water of other marshes was drunk without this effect. If the morbific power were simply the product of chemical decomposition, this exclusiveness would be entirely inexplicable. I have no hesitation in saying decidedly that marsh miasm—malaria—must consist of low vegetable organisms, whose development is chiefly due to the putrefaction of vegetable substances. It is true these low organisms have not actually been observed. No one has seen "malaria spores," but the facts above mentioned, as well as many other causes, urge us to believe that the poison exhaled by marshes, as well as that given off by a patient with measles, is an organic living substance. There is, however, an important difference between the miasma vivum, which is the specific cause of intermittent fever, and the contagium vivum by which the acute exanthemata, exanthematic typhus, and other infectious diseases, spread. The latter reproduces itself in organisms infected with it; malaria, on the other hand, is not reproduced in the body of a patient with intermittent fever. There is no soil in the human body favorable to its development or increase. Intermittent fever is never introduced into other places by patients who have caught it in a swampy region. While a short residence in a malarious district is often enough to give intermittent fever, one may share the same ward with a large number of intermittent patients in a hospital, at some distance from such a place, without danger. Hence, in distinction to the "contagious" diseases, malarial fever is termed "miasmatic contagious" or "purely miasmatic."

There are extensive sections of country where the circumstances for the formation of malaria exist everywhere, in all parts of which intermittent fever occurs; but there are also small circumscribed malarial foci, where numerous cases of intermittent are seen every spring and summer, while the whole surrounding country remains free. In these last-mentioned circumscribed malarial foci, in towns lying near marshes, in certain sections or streets of cities, where intermittent fever is endemic, some very interesting observations have been made about the extension of malaria: among other things, it has been shown that, from its point of origin, miasm spreads more readily in a horizontal than in a vertical direction, it is often arrested by insignificant obstacles, such as groves, stone walls, etc., and rarely passes the boundaries thus formed, unless the wind be in a particular direction.

The extensive epidemics of intermittent fever which occasionally appear are very remarkable. During such epidemics, while the cases are unusually frequent in places where the disease is endemic, they also occur in places where either no cases or only sporadic ones had been seen for years. These epidemics do not always come in very hot, moist seasons, so that they might be referred to the circumstances

being very favorable to decomposition of vegetable substances, and hence uncommonly favorable for the development of malaria, and for its spread from places where it always exists to others that are generally free from it; but they appear to be due to other unknown causes, which also favor the development of malaria, or to an extension of the disease from its point of origin to distant places by currents of air. We do not know why, but great epidemics of intermittent have often preceded epidemics of Asiatic cholera. In hot countries, cholera and intermittent, and, still oftener, dysentery and intermittent (see next chapter) frequently prevail at the same time.

The *sporadic* occurrence of intermittent is most curious. Those cases where persons affected with malaria move from places where malaria is prevalent, and continue to present the symptoms of infection for a time after settling in their new home, are not to be counted as sporadic. But, both in city and country, cases occur where the persons could not have been infected elsewhere. There is no explanation for these cases, except the supposition of very limited foci, which are harmless for persons outside of them, or of a very diluted miasm, which only affects a few persons who are peculiarly disposed. There is not the least ground for the supposition that, under some circumstances, intermittent may result from other causes than from infection with malaria.

Every age, both sexes, and all constitutions, have a predisposition to intermittent fever, and the greater or less tendency to the disease, in various persons, does not depend on the constitutional differences to which it has been referred, but to other only partially understood individual peculiarities. Exhausting exercises, and other debilitating influences, errors of diet, and particularly catching cold, increase the predisposition so much, that persons, who have long been exposed to malaria with impunity, are not affected by it till one of these causes has acted on them. The same explanation must answer for those cases where persons who have escaped the disease in malarious places are attacked by it after leaving them. Then the infection has evi dently taken place earlier, but has not shown its effects in the person who was little disposed to the disease, and has not caused injurious results till the necessary predisposition was induced by other noxious agents. Among the influences that increase the predisposition to intermittent, the most evident is one or more previous attacks of the disease, which is just the opposite of the case in the acute exanthemata and typhus. There is only an accommodation of the organism to the poison to the extent that persons who have been exposed tc it for some time have a chronic disease with enlargement of the spleen, without fever, instead of having intermittent fever. The

geographical extension of intermittent fever is immense; in the torrid zones the disease is especially frequent, and it prevails in most places that are not very dry: in the temperate zones it chiefly affects certain more or less extensive districts; in the frigid zones it does not occur. For further information, we refer to *Griesinger's* work and to the very exhaustive treatise of *Hirsch* on the geography of intermittent fever.

Anatomical Appearances.—Autopsies of persons who have died of simple intermittent fever are rare. They show no characteristic changes beyond those that we have already described (Vol. I., Diseases of the Spleen). If simple intermittent fever depends on infection with malaria, the anomalies of the blood dependent on the infection have so far escaped chemical and microscopical examination. The impoverishment of the blood in red corpuscles and albumen, which always results from long-continued intermittent, depends on consumption from high fever, and perhaps also on degeneration of the spleen. On autopsy of persons who have died of pernicious intermittent, we often find signs of melanæmia and occasionally remains of inflammation, effusions of blood, or excessive hyperæmia in different organs.

Symptoms and Course.—I. *Simple Intermittent Fever.*—The period of incubation of the malarial infection is not exactly known. It is probable that, at most, two weeks may pass from exposure to the miasm till the first symptoms appear; in some cases the latter seem to follow the former immediately.

Before the intermittent character of the disease, to which it owes its name, becomes very evident, before attacks of fever (paroxysms) and intervals of exemption from fever (apyrexias) alternate, the infection with malarial poison not unfrequently evinces itself by a continued, only more or less remitting and exacerbating, disturbance of the general health and various functions. This constitutional disturbance, which is called the *prodromal* stage of intermittent, has no characteristic peculiarities, and can only be rightly interpreted and distinguished from the prodromal stage of other infectious diseases by bearing in mind the endemic and epidemic circumstances. After the prodromal symptoms have lasted several days, or in other cases without their appearance, the first fever paroxysm occurs.

A paroxysm of intermittent fever consists of three stages: chill, fever, and sweating. The chill begins with a fit of weakness, and great faintness; the patients gape, and stretch their limbs. These symptoms are soon accompanied by a subjective sensation of cold, at first consisting of cold shiverings over the skin; afterward, of continued chilliness, so that the patient wants to wrap up as warmly as possible. When the chill increases, the limbs tremble involuntarily, the lips quiver, the teeth chatter, and the whole body is often shaken around

in bed. When the chill begins, there is also more or less headache, oppression of the chest, and hurried respiration; the latter, and the quivering of the lips, render speech indistinct and interrupted; there is often vomiting also, especially if the chill comes soon after eating. Objective examination first shows the altered appearance of the patient; he looks like a person exposed to severe cold, without sufficient clothing, and who is consequently thoroughly chilled. The size of the body appears diminished; the face is sunken; the nose pointed, the rings are too large for the fingers. As the flow of arterial blood to the skin is impeded, the latter appears pale, and, as the blood collects in the veins and capillaries, the lips, ends of the fingers and toes, look blue. The flow of blood to the fingers is often entirely arrested; they look waxy, have no feeling, and do not bleed if wounded. The skin of the extremities and body has the appearance known as goose-flesh (cutis anserina). The pulse is very frequent, small, and hard; the secretion of urine is usually increased; the urine passed is limpid, and of low specific gravity. Physical examination generally shows enlargement of the spleen. While the diminished supply of warm blood during the chill causes the periphery of the body to assume more and more the temperature of the surrounding atmosphere, and to actually sink several degrees, the temperature of the blood and internal organs rises rapidly. This rise, which begins even before the chill, reaches two or three degrees, and, in severe cases, still more. The above symptoms are mostly to be ascribed to spasmodic contraction of the muscles of the skin and peripheral arteries during the cold stage. The immediate results of this spasm are the goose-flesh, the small, hard pulse, and the deadness of the fingers. The contraction of the muscles of the skin and peripheral blood-vessels secondarily induces the apparent diminution in size, the dryness and pallid appearance of the skin, the collection of blood in the veins, and the fall of temperature on the surface of the body. The chilly feeling of the patient is too decided to be referred exclusively to the fall of temperature of the periphery; on the other hand, the continued coolness of the surface, even under thick feather-beds (which is peculiarly remarkable, because the body gives off but little heat to the parts around), is certainly due to the fact that, during the chill, there is less warmth supplied to the surface, on account of the diminished supply of blood. Lastly, the disturbance of the peripheral circulation increases the lateral pressure in the vessels of the internal organs. But, as we do not know whether the spasm of the muscles of the vessels is limited to the peripheral arteries, and does not extend to the internal ones, it is doubtful whether the headache, oppression, accelerated respiration, vomiting, increased secretion of urine, swelling of

the spleen, etc., can be referred to collateral fluxion to the organs implicated in these symptoms. At all events, as we have before said, collateral fluxion to the spleen is not the sole cause of enlargement of that organ. The cold stage lasts from half an hour to three hours; in the first paroxysms, it is usually shorter and less severe than subsequently; and its intensity and duration generally decrease again if the disease last a long time. The hot stage does not begin suddenly, but gradually; at first the chill is only interrupted by temporary flashes of heat, and a permanent feeling of warmth only comes on by degrees. The headache becomes more severe; the patients grow restless, and not unfrequently slightly delirious or somewhat stupid; the feeling of oppression on the chest increases, while the breathing almost always becomes deeper, freer, and slower. Thirst is greatly increased. The appearance of the patient changes. The size of the body apparently increases, the color returns to the skin, the pale, livid look, the goose-flesh, the cyanotic hue of the lips and ends of the fingers disappear; the face becomes flushed, and herpetic vesicles often come out on the lips; the previously small pulse grows full and strong, the carotids throb, the urine becomes more saturated, and the enlargement of the spleen increases. In the hot stage, the bodily temperature is increased at the periphery also; at the commencement of this stage, the temperature of the blood reaches its highest point, and remains there till toward its termination, when it gradually begins to fall. All these symptoms show that the spasm in the muscles of the skin and vessels has disappeared and given place to a subparalytic state. The relaxation of the cutaneous tissue and of the walls of the vessels sufficiently explains the increased entrance of blood into the previously bloodless tissues, and their consequently increased color and warmth. The severe headache, the oppression of the chest, which are often accompanied by catarrhal symptoms, and finally the continued enlargement of the spleen, render it very probable that the arteries of the brain, bronchi, and spleen, are in the same condition as those of the periphery. The duration of the hot stage varies; sometimes it is only a few hours, in severe cases the dry heat continues six, eight, or even twelve hours before a pleasant perspiration breaks out on the skin. The sweating stage begins with moisture in the armpits and on the forehead; but soon the perspiration, at first moderate, but later very copious, extends all over the body. The patients feel greatly relieved, the headache remits and gradually disappears, the mind becomes clear, the oppression passes off, the depth and frequence of the respiration become normal, the thirst is less annoying, the pulse is full, soft, and less frequent. The dark urine generally deposits rich sediments of urates; this depends on the great concentration resulting from the loss

of water by evaporation and sweating; it does not occur if the patients replace the loss of water by drinking freely. During the sweating stage, the bodily temperature gradually falls, and toward its end becomes nearly normal. When the paroxysm is over, and the apyrexia begins, most patients fall into a deep sleep, from which they awake much fatigued and dull, it is true, but feeling relatively well. When all the stages of the paroxysm are well marked, the disease is called an intermittens completa; if one or other stage fails or is indistinct, it is called incompleta. In rare cases, the stages of the fever are said not to follow the regular order; for instance, the cold stage closes the paroxysm instead of opening it (typus inversus).

After the first paroxysms of intermittent, the apyrexia is rarely "pure." The patients have no fever, it is true; in some cases the temperature is even low and the pulse slow; but the appetite is often poor; the tongue coated, digestion disturbed; the patients are sensitive to changes of temperature, and complain of an indefinite feeling of illness. After a few paroxysms the disturbances usually subside, and during the apyrexia the only symptoms we notice are steadily-increasing debility and more evident impoverishment of the blood. If intermittent fever is very protracted, the apyrexias often become impure again, and the intermittent type approaches the remittent (febris intermittens subcontinua).

According to the rhythm in which the paroxysms follow each other, we distinguish intermittens quotidiana, tertiana, and quartana. In the quotidian, the paroxysms recur in nearly or exactly twenty-four hours; in the tertian, in twice twenty-four hours; in the quartan, in three times twenty-four hours after the commencement of the last one. It is less certain if there be also a five, six, or seven day rhythm, or if the intervals be even longer. The most frequent varieties are the tertian and quotidian. If the paroxysms recur at an earlier hour than it did the last fever-day, the disease is called *anticipating*, in the opposite case it is called *retarding*. By continued anticipation or retardation, the rhythm of the fever may change, and a tertian may become a quotidian, or the reverse. If a paroxysm last almost till the commencement of the next one, we have a *febris intermittens subintrans*. When the apyrexia of a quotidian, tertian, or quartan intermittent is interrupted by a weak, short paroxysm, which often occurs at a different time of day, it is called *febris intermittens duplicata*. In *quotidiana duplicata* there are two attacks each day, one severe, the other mild; in *tertiana duplicata* there is one attack each day, but one day it is mild, the next weak, etc.; in *quartan duplicata*, paroxysms occur on two successive days, while the third day remains free. Intermittent fever never begins double; this rhythm usually develops

after the disease has lasted a long time, or particularly after numerous relapses.

The course of intermittent is so often modified by the administration of medicines, especially of quinine, that few physicians have actually observed a case left to itself, or, as is generally said, the normal course of an intermittent. On this point also we are indebted to the homœopaths for some important information. In the first place, there is no doubt that the peculiar fever paroxysms and apyrexias, which characterize infection with malaria, almost always continue for a time, even if the patient be removed from further action of the poison. Among other cases, I know of a young sailor who suffered from intermittent fever in Greifswald, and who had a relapse while at sea, four weeks after his last attack. It is also certain that, when a long time has passed since the action of the poison, the symptoms of malarial poisoning almost always disappear without medical aid. To avoid illusion, we must bear in mind that it is not only those who fly from the malarious region after acquiring intermittent fever, but also those who remain in the place till the malaria has disappeared, that finally escape the action of the poison. At a time when no new cases are observed in a place which has been visited by intermittent, it would be foolish to regard the final cure of a case, which had previously been treated ineffectually, as a proof of the peculiar efficacy of the remedy last used. As the prevalence of the malaria among us only lasts a few months, the number of spontaneous cures is certainly much greater than is usually supposed.

Another question is, whether, from continued action of the poison, the organism becomes so blunted to its injurious influence, that the symptoms of intoxication cease. At all events, such cases are rare, and it is more frequent to find those where continued action of the malaria has only modified the symptoms of intoxication, so as to change a pure intermittent into an intermittens subcontinua, or chronic malarial illness without fever. As was stated, when speaking of the individual paroxysms and apyrexias, after a prodromal stage but little characteristic, most cases of intermittent begin as tertian or quartan fevers, the apyrexias being at first imperfect, but becoming pure after a while. When the disease has lasted even a very short time, the patient appears very pale and cachectic, and this symptom is the earlier and the more marked, the greater the enlargement of the spleen. If the spleen remain small, from rigidity of its capsule, or from other unknown causes, the patient does not have this peculiar pale and sickly appearance until later. From this fact, which may be observed in most cases, as well as from the second fact, that the healthy color quickly returns when we can rapidly reduce the

size of the spleen, we have concluded that the great pallor of the patients after the *first* attacks of intermittent does not solely, and, at all events, does not exclusively, depend on general impoverishment of the blood, but mostly on the lack of blood in the skin, which must be induced by its collection in the spleen. While *Junod's* boot is applied to one extremity, there is great hyperæmia of that limb, and consequent anæmia of the rest of the body; if the boot be removed, the normal distribution of blood is soon reëstablished. We do not, however, mean to say that the fever of intermittent differs from other fevers in regard to the consumption of the body; on the contrary, intermittent fever would be one of the most dangerous of diseases, if those inexplicable pauses did not occur between the paroxysms, during which the consumption is interrupted, and the tissues of the body restored by plentiful supply of nourishment, more than in any other feverish disease. From the high temperature (106° Fahr. or more), reached in intermittent fever, the consumption of the body is very marked. This is evident, not only from the increased production of urea, but also from the emaciation and hydræmia that occur in protracted cases. There is no doubt that the *production* of urea is only increased during the paroxysm, and not during the apyrexia; on the other hand, in two cases of quartan intermittent, I found that the *excretion* of urea was increased during the apyrexia also, or, in other words, that the increased amount of urea produced during the paroxysm was not all excreted till some time during the following apyrexia. If the paroxysms be not temporarily or permanently arrested by the administration of quinine, or if they do not soon cease spontaneously, the hydræmia at length becomes so excessive, that in many cases more or less dropsy occurs, without the urine being diminished in quantity, or containing albumen. This dropsy is analogous to those occurring during other exhausting diseases, although it is probable that the organic and functional disturbances of the spleen accompanying protracted intermittent fever add to the degree of hydræmia, and to the frequency of the dropsy. The longer the affection lasts, the more reason there is to fear the development of permanent organic change in the spleen, liver, or kidneys: this usually assumes the form of lardaceous degeneration, with coincident deposit of pigment, and leads to incurable disease. Continued intermittent fever often induces chronic parenchymatous nephritis also, and, in other cases, a hæmorrhagic diathesis. These severe sequelæ of simple intermittent fever are generally prevented by proper treatment; and, even where intense malaria is endemic, under careful management, excessive dropsy, fatty degeneration of the abdominal organs, and cachexia, rarely occur. It is true, neither quinine, nor any other known remedy is so certain an antidote for malarial poison as

to cut short the entire disease; but, for the most important and dangerous symptom, the fever, quinine is almost infallible. After giving proper doses of quinine, the patient almost always escapes a series of paroxysms; and, as the cachexia and impoverishment of the blood, and, to some extent, the spleen-affection also, probably depend on the fever, the patients not only do not grow worse during the administration of the quinine, but they improve and pick up even if the disease be not entirely extinct. Although, for the sake of brevity, we may designate as relapses the many cases where paroxysms recur shortly after stopping the quinine, strictly speaking, it is incorrect; for the cases where these so-called relapses occur, even after moving into a region free of malaria, prove that the disease was not cured, but only one of its symptoms removed. The homœopaths assert that under their treatment there are no relapses: there is some truth in this assertion; for, when the paroxysms have ceased under homœopathic treatment, the disease is certainly all gone. The fact, that, after the administration of quinine in sufficient doses, relapse does not occur in many cases, rather favors the view that, besides its palliative action on the paroxysms, this medicine has also a favorable influence on the entire disease caused by the malarial infection; but it is also possible that in such cases the palliative action continues till the disease passes off spontaneously. When the action of quinine is only palliative (as it is in the majority of cases), usually about seven, fourteen, or twenty-one paroxysms are missed, and the next one occurs in about two, three, or four weeks, rarely sooner. According to my own observation, the assertion, that the relapses almost regularly take place the fourteenth, twenty-first, or twenty-eighth day, is exaggerated; I have far more frequently seen relapses a few days before or after these dates. It is not at all rare for relapses to occur three or four times, or oftener, and for the disease to run on interruptedly for months, before actually ceasing. I deem it proper to speak of the modification of the course of intermittent fever by quinine under symptomatology, because, as we have already said, most physicians have no opportunity of observing intermittent fever without this modification of its course. Much that is said in the text-books on medicine, about the course of intermittent fever, certainly does not refer to the disease when left to itself.

II. *Pernicious Intermittent Fever.*—Intermittent fever may prove dangerous to children, to the aged, and to very debilitated or sickly persons, without being of unusual intensity or duration, or having any complications. Children inclined to convulsions not unfrequently have an epileptiform attack during the cold stage, as they also have in the initiative chill of inflammatory diseases. This is not usually danger-

ous, but occasionally it passes into general paralysis, ending in death. In very old or broken-down persons, the danger from a simple paroxysm lies chiefly in the threatened exhaustion of the still remaining strength. Such cases, which only take on an acute course from individual peculiarities of the patient, are not usually called pernicious.

Some cases of intermittent acquire a pernicious character, from the usual symptoms becoming very intense, or being protracted a long time. In this class belong the cases where the hyperæmic swelling of the spleen is so great that its capsule is ruptured, or where the chill is so severe as to cause dangerous obstruction of the circulation, as well as those cases where the paroxysms do not cease in from six to ten hours, but continue for twenty-four hours, or longer, and leave the patient very much exhausted, or where only certain stages continue beyond the usual time. It seems to me very probable that some forms of the so-called pernicious fever also should come under this head, as it is probable that they are not complicated, only that the customary symptoms are greatly increased. Even in simple normal intermittent there is an increase of the temperature, such as is observed in scarcely any other disease; and the short time that this increase lasts is the only reason that it does not prove dangerous to the patient. In all diseases where the bodily temperature becomes excessive, or remains high for a long while, we have the symptoms of great adynamia, and finally of paralysis of the heart, and these are the most prominent symptoms of pernicious fever. The deep stupor of patients with *febris intermittens comatosa*, as well as the delirium preceding the coma, reminds us strongly of the symptoms in severe typhus, malignant measles, and other infectious diseases accompanied by high fever. When these cases terminate fatally, we do not generally find any anatomical changes in the brain. In *febris algida* the heart's action grows feebler, the pulse smaller; the blood collects in the veins; there is cyanosis; the temperature of the periphery closely approaches that of the surrounding air, because the loss of warmth due to the obstructed circulation is not replaced by a supply of warm blood; the patients grow cool; in short, we have the symptoms of acute paralysis of the heart, which may even result from the intensity of the fever without complications. There is no doubt that, in these severe forms of intermittent fever, there is often pigment in the blood; but the coincidence of melanæmia with severity of intermittent fever does not justify us in regarding the latter as a result of the former. On autopsy of several cases of intermittent fever that died with severe brain-symptoms, *Frerichs* found melanæmia, it is true, but he found no pigment in the capillaries of the brain; and I have observed similar cases.

Under the head of congestive, pernicious intermittent, in the strict sense, we must class those cases where the malignant course of the disease is caused by hyperæmia, effusions of blood, inflammations, and, perhaps, also by obstructions of the circulation in different organs, induced by stoppage of the capillaries with pigment. Probably these disturbances of nutrition and circulation in the central organs of the nervous system are the origin of the maniacal, apoplectic, epileptiform, and tetanic convulsions, which occasionally complicate the paroxysms, and have led to the designations febris intermittens perniciosa, maniacalis, apoplectica, epileptica, tetanica. A rather frequent but rarely dangerous complication of intermittent fever is a severe bronchitis, which exacerbates with every paroxysm and remits with each apyrexia. Febres intermittentes comitatæ pneumoniacæ and pleuriticæ, or cases of intermittent hæmoptysis, are rare. In many cases of congestive intermittent, there is jaundice. Copious watery transudations from the intestinal canal, violent vomiting, and profuse diarrhœa, may cause thickening of the blood, and give the paroxysm a great resemblance to the algid stage of cholera. In some cases there are also profuse intermittent intestinal hæmorrhages. (Among fifty-one cases of pernicious intermittent, observed by *Frerichs*, there was profuse diarrhœa seventeen times, profuse intestinal hæmorrhage three times.) The serous transudations and hæmorrhages into the intestinal canal may depend on acute congestion due to obstruction of the hepatic capillaries by pigment, but this has not been proved in all cases. Lastly, we have to mention the complications of intermittent with diseases of the kidneys. They evince themselves by albuminuria, hæmaturia, and in severer cases by the suppression of urine. (In the fifty-one cases of *Frerichs*, albuminuria was seen twenty times, suppression of urine five times.)

Pernicious intermittent fever occurs chiefly in tropical-fever regions, it is true, but even with us, in places where intense malaria prevails, it is not rare, and individual cases are seen in every large epidemic of intermittent fever. The malignant character either appears at the start, or does not manifest itself till the second or third paroxysm, or even later. After malignant congestive symptoms have appeared, the apyrexias are often so imperfect that the diagnosis is very difficult or even impossible. In the epidemics observed by *Frerichs*, most patients were sent into the hospital with the diagnosis of a typhus.

III. *Concealed Intermittent Fever.*—While, as a rule, intermittent fever results from infection with malaria, we not unfrequently see exceptional cases where, instead of fever-paroxysms, there is neuralgia, the attacks of neuralgia being separated by regular intermissions, corresponding to the apyrexias of a simple intermittent. These deviations from the rule are inexplicable, it is true, but this is also true of regular

simple intermittent fever, which we do not in the least understand. The intermittent neuralgiæ resulting from malaria, which chiefly bear the name of intermittentes larvatæ, most frequently affect the supra-orbital, more rarely other branches of the trigeminus or other nerves. We may omit a description of the attacks, as they are about the same as those of neuralgic attacks from other causes. They are accompanied by a slight elevation of temperature, perceptible by the thermometer. In rare cases, other anomalies of excitement of the cerebro-spinal and vaso-motor nerves appear to result from malaria. Among these are intermittent anæsthesia, paralysis, spasms, psychical disturbances, and hyperæmia or œdema of different organs.

Treatment.—Prophylaxis requires that the State should attempt to remove by sanitary laws (which do not come in our province) the injurious influences which notoriously favor the development of malaria; that it should drain the marshes in whose vicinity intermittent fever is endemic, and should protect by dikes those lands that are overflowed yearly. Moreover, persons obliged to reside temporarily or permanently in malarial regions should observe certain rules, which afford more protection than any prophylactic medicines. *Hauschka*, who has apparently had great experience, gives very precise and practical rules for life in malarial regions. In his special pathology and therapeutics, he advises: 1, on going to a dangerous place, to assume at once the peculiar mode of life of the inhabitants; on the Weichsel to drink schnaps, in Banate slivovitz, in Hungary only to eat melons and pickles with plenty of pepper, in Italy to drink plenty of lemonade and black coffee, and avoid eating at night, also to drink water prepared as is customary at different places; 2, to suit the clothing to the temperature of the time of day, especially to protect the person by warmer clothes against the cool evening air, and to keep the windows closed at night; 3, to seek a dwelling high and dry above the marshes, etc.; 4, to avoid getting wet through, errors of diet and other excesses, so as not to bring on any other disease by which the tendency to malaria will be increased; 5, to avoid certain things, such as the use of vegetables, milk, and bathing in rivers or the sea, especially after sundown. Sleeping in the open air seems to be most dangerous.

Treatment of the paroxysm may almost always be dispensed with, except as regards directing the regimen. As soon as the first symptoms of chill are observed, the patient should go to bed; but we should warn the attendants against piling on too many bed-clothes, as they do not warm the skin, and only interfere still more with the circulation in the peripheral parts, and with the impeded respiration. If the chill be very severe, the skin may be rubbed with warm woollen cloths, and warm bottles placed in the bed. Hot drinks neither re-

lieve the feeling of chilliness nor increase the temperature in the peripheral parts, which is diminished in spite of the increased temperature of the blood; but we may yield to the generally urgent entreaties of the patient for warm drink, so far as to allow him a few cups of tea, if we are certain that he will be moderate. If there be severe vomiting during the cold stage, we may give effervescing powder, and, if this fail, a few drops of laudanum. If the patient become so collapsed as to alarm us, we may give analeptics, strong coffee, wine, camphor, ether, and opium, have the patient rubbed, and apply sinapisms. In the hot stage we may try cooling treatment, light covering, plenty of cold drink in small quantities; where there is severe congestion of the brain, use cold water or ice-compresses to the head, and sometimes local blood-letting. Venesection is only to be practised in the most urgent cases, as it rarely does good, and may cause dangerous collapse; in its place, in attacks of pernicious fever, besides local blood-letting and derivatives, the use of opium has proved the most efficient treatment. The sweating stage must be awaited in bed. Especial treatment to increase the sweating is unnecessary, as there is no advantage from its being very profuse. The patients should not change their underclothes till this stage has terminated. The treatment during the apyrexia aims at preventing the return of paroxysms by the administration of quinine, unless there be some peculiar objection to giving that medicine. The formerly common idea, that it was advantageous for the patient to have a certain number of paroxysms, and that the fever should not be arrested till after the third, fifth, or even seventh attack, was based on false premises. The sooner he is freed of his paroxysms the better for him. A morbid state of the gastric mucous membrane, however, which occasionally occurs after the first paroxysm of intermittent, contraindicates the administration of sulphate of quinine, and, in mild cases at least, this morbid state should be removed before ordering the irritating salt. In treating intermittent fever, the diagnosis of gastric catarrh, or foul stomach, is not made very exactly, and emetics are used too freely; some physicians begin the treatment with an emetic, as a matter of course; others prescribe muriate of ammonia till the tongue is thickly coated, and then give an emetic to prepare the patient for quinine. Unless certain indications, that we have given for the administration of emetics in gastric catarrh, be present, we consider it improper, or at least superfluous, to open the treatment of intermittent with one. But if, at the commencement of the fever, the patient has made great errors of diet, especially if, just before the paroxysm, he has filled his stomach with food which has remained undigested, and by decomposing has irritated the gastric mucous membrane, there is just cause for giving an emetic. We have

already described the difference between gastric catarrh due to indigestion and the slight dyspepsia accompanying most fevers which requires no emetic, and does not prevent our giving quinine. *Pfeufer's* recommendation, to give quinine in one large dose instead of in several smaller ones during the apyrexia, has very rightly become popular. Usually, in adults, one dose of ten grains of sulphate of quinine suffices to arrest the chills; in children, five grains suffice, in still smaller children, two or three grains answer. Another slight chill often occurs, and the attacks then cease. It is best to give this dose of quinine some hours before the expected chill, so that we may be sure of its acting during the apyrexia. Small doses should only be preferred when large ones are vomited by the patient; in such cases we may give two grains every two hours, till twelve or fifteen grains have been taken during the apyrexia. Quinine is most frequently given in powder or pill (quiniæ sulphat. gr. x, ext. gentian. q. s. ut f. pill. no. iv). Solution of bisulphate of quinine (quiniæ sulph. gr. x, aquæ meliss. ℥ iij, acid. sulphur. dilut. ℈ ss, syrup. ℥ ss) is said to be particularly effective, but it is unpleasant to take, especially in large doses. If quinine is not borne by the stomach, it may be given by injection, as it acts just as well by the rectum (or by hypodermic injection). Other salts of quinine have no advantage over the sulphate; quinoidine and tincture of quinoidine are cheaper, and hence may be preferably used in poor practice; of the latter remedy we may give twelve or fifteen drops every two hours, or give a teaspoonful a short time before the expected chill. According to *Wunderlich*, this is quite as efficacious as quinine. If the patient has escaped one chill under the above treatment, he almost always escapes a second and third, or even a number; but it is advisable to continue small doses of quinine for a few days longer, unless the spleen has distinctly and steadily decreased in size. The more perfectly the spleen returns to its normal bulk, the greater is the probability that there will be no relapse. I have already given my ideas about the custom of repeating the quinine on the seventh, fourteenth, twenty-first, and twenty-eighth days. When this is being done, the chill often occurs on the day just preceding the one when the medicine was to be given. I think it much safer to tell the patient that he will probably have a relapse toward the end of the second, third, or fourth week, and that at these times he must watch carefully for any premonitory symptoms, and on the slightest suspicion of a chill he must take a full dose of quinine. By careful attention to the rudimentary attacks which almost always precede perfect relapses, most patients of moderate intelligence may protect themselves from the latter. The belief, that, in cases of relapse, the patient must daily take an ounce of tincture of Peruvian bark in-

stead of quinine, is antiquated. Besides the administration of quinine or its repetition, we should urge the patient to adopt all the rules spoken of under prophylaxis. When the circumstances of the patients permit, we should have them leave the affected place for six or eight weeks. We have already explained that this does not render the administration of quinine superfluous.

If chronic malarial dyscrasia develop, besides a nutritious and strengthening diet, the patient should twice a day take half a glass of bark and wine (tinc. cinchonæ ʒ i, tinc. cinnamom. ʒ i, acid. sulph. aromat. ʒ ij to a bottle of Rhine wine), or three or four teaspoonfuls daily of tinc. cinchonæ comp., combined with large doses of iron. In most cases the benefit of this treatment is very evident: even advanced dropsy usually disappears in a short time without any diuretic remedies; should it unexpectedly fail, we ought to absolutely insist on a temporary change of residence, even if it be very inconvenient.

The above treatment is almost always successful in simple intermittent. Experiments with substitutes for quinine, such as salacin, piperin, salt, etc., have led to negative or doubtful results. The only febrifuge, except Peruvian bark and its preparations, that deserves confidence, is arsenic, in the form of Fowler's solution (four to six drops three times daily during the apyrexia). In view of the far more certain and safe action of quinine, I consider the use of arsenic as only justifiable in those rare cases where quinine fails, even in large doses.

In pernicious intermittent, our first object is to prevent the occurrence of the next attack. While carrying out the treatment required by the congestive symptoms during the attack, we should not wait for a complete intermission before giving quinine; but, as soon as there is the slightest abatement of the symptoms, we should give large doses of quinine (ʒ j to ʒ ss. or more), and, if the patients cannot swallow, give it by enema [or, better, by hypodermic injection, giving about one-third the dose by the mouth, *perfectly* dissolved].

In the concealed form of intermittent, quinine is almost as efficacious as in the simple form; but even here its action appears only palliative and symptomatic, as it does not prevent relapses.

CHAPTER XII.

REMITTENT AND CONTINUED MALARIAL FEVER.

Etiology.—Remittent malarial fevers occur chiefly in the tropics, it is true, but they are occasionally observed among us also, in places where malarial diseases are endemic, and at other places also when epidemics of intermittent are prevailing. Hence I cannot class remit-

tent malarial fever among the exotic diseases and pass it by, but I shall follow the excellent description of *Griesinger* in my short account of it, as I have but little knowledge of the affection from personal observation.

The dependence of remittent fever on malarial infection is shown, first, by its exclusive occurrence in places where we know, from the quality of the soil and climate, and from the occurrence of numerous cases of intermittent, that there is an intense malaria; secondly, from the many cases where remittent fever becomes intermittent. We do not know whether the change of symptoms be due to a modification of the poison, and, if so, in what this modification consists. The more severe the cases of intermittent, the more frequent are the remittent cases.

Anatomical Appearances.—As regards the melanæmia, the *post-mortem* appearances of remittent fever correspond with those of pernicious intermittent. At least, in almost all places where remittent fever is endemic, in the rare cases of autopsy it has been noticed that the cortical substance of the brain is of a dark, ashy hue, and the spleen and liver are blackish. Besides these changes, which are not constant, traces of more or less severe icterus are often found, sometimes with, again without, obstruction of the gall-ducts; as well as the remains of catarrhal and diphtheritic inflammation of the intestines, and, more rarely, hæmorrhages into the stomach, hæmorrhagic infarctions in the lungs, or lobular pneumonia.

Symptoms and Course.—*Griesinger* distinguishes three forms of remittent fever. According to him, the first and *mildest* form begins with a sudden feeling of severe illness, high fever, and foul stomach. These are soon accompanied by enlargement of the spleen, mild jaundice, irregularity of the bowels, discoloration of the fæces, and herpes labialis. The depression of the patient, the pain in the head and limbs, the dizziness, noise in the ears, and not unfrequent bronchitis, remind us of a commencing typhus; but from the first the fever shows a decided remittent character. Irregular exacerbations, which subsequently become regular, and usually have a quotidian type, are followed by distinct remissions. These gradually pass into perfect intermissions, the patient sweats freely, and feels well; the remittent becomes a simple intermittent. In other cases, even without this change, the disease terminates in recovery, by a gradual decrease of the symptoms; it lasts from a few days to three weeks.

In the *severer* form the fever is very high, the remissions are well marked only at first; the disease reminds us of a severe typhus; the patients become stupid and delirious; the tongue grows dry, and the spleen is enlarged. Generally, also, but not always, there is icterus;

other cases are complicated with pneumonia, dysentery, etc., and still others have indications of the so-called pernicious attacks. The disease usually lasts from eight to fourteen days. If it ends in recovery, it usually first becomes intermittent. Death may occur suddenly with the symptoms of a pernicious intermittent.

The *severest* forms run their course with the symptoms of an indistinct and irregular exacerbating and remitting fever of very adynamic character. The patients collapse quickly, and soon fall into deep apathy. There are also various but not constant disturbances of function and nutrition in almost all the organs, so that the symptoms of the disease vary greatly. Many patients become jaundiced. There is often epistaxis, vomiting of blood, and hæmaturia; in other cases there is albuminuria, or suppression of urine; in others, symptoms of cholera or dysentery; the spleen and liver enlarge considerably, and often become the seats of inflammation and suppuration. Inflammatory exudations not unfrequently form in the serous membranes and lungs also, and in the skin there are petechiæ, bed-sores, and gangrene. Death usually occurs in this stage, with coma or convulsions, or with the symptoms of febris algida.

Treatment.—According to *Griesinger*, in the milder forms of remittent fever, the acute affection of the gastro-intestinal mucous membrane should be combated by absolute diet, acids, and, if requisite, by emetics and laxative medicines. We should give quinine as soon as the remissions and exacerbations become decided, and the latter begin with a chill. Any complications should be treated separately, as quinine alone does not answer for them. In the severer and severest forms, the most important indication is the early and continued use of quinine in large doses, till improvement begins. At the same time, the congestive symptoms are to be treated symptomatically, just as in pernicious intermittent fever.

[At present malarial poison is by many supposed to consist of microscopic vegetable parasites, in algæ cells and spores, from the decomposition of vegetable substances. *Harkness* asserts that the algæ cells, which *Salisbury* found in the sputa of patients and regarded as the active principle of malaria, are found not only in malarial regions, but on the highest Alps, and occur in the sputa and other secretions of healthy persons.

Intermittent fever is a wide-spread disease. It is especially frequent in the tropics at places which are not particularly dry; in the temperate zones it occurs at certain places; but the frigid zones escape. It is very common in Western Africa, the West Indies, Mexico, and our Southern States, but occurs to some extent in all the States.

The period of incubation for malaria is uncertain. Patients subjected to the poison for a long time, or suffering from the disease, look pale and cachectic, probably from changes in the blood, perhaps partly due to fever, partly to affection of the blood-forming organs. The red blood-corpuscles are less numerous; the remains of those destroyed appear as pigment in the blood and some of the organs. This diminution in number is greatest at first, but goes on till recovery begins; but at the same time it is said they increase in size. *Kelsch* says the white corpuscles decrease in number faster than the red do, but in the interval increase gradually, and that at the time they are least numerous the spleen is largest.

If after the intermittent paroxysms have ceased the spleen or liver remain enlarged, the patient cannot be regarded as cured. Chronic enlargements of the spleen may attain an enormous size; or there may be amyloid degeneration of the spleen, the liver, or the kidneys.

Where in the treatment of malarial diseases the preparations of cinchona prove ineffectual, we may try eucalyptus globulus, of which we may give ʒ ss–ʒ ij of the tincture, or 10–20 drops of fluid extract, several times daily. Another substitute for quinine is arsenic, either as pills of arsenious acid or as Fowler's solution. In cases of malarial neuralgia, or where there is gastric disturbance, the Fowler's solution may be used hypodermically. Faradization of the splenic region is said often to diminish the size of the spleen, as also the use of cold douches over that organ while the patient is in a tepid bath.]

[CHAPTER XIII.

YELLOW FEVER.

Without discussing the question as to when and where yellow fever originated, we may say that in the United States its different epidemics seem to come from the West Indies, and to start among us from New Orleans; thence its course of travel is along rivers or by the seaports.

The cause of the disease is not certainly known, but it is supposed to be in the nature of germs. The affection is not contagious; i. e., a patient having it, if stripped of his clothing and disinfected, may be placed among other patients without spreading the disease. So, the germs do not develop and multiply *in* the

patient, but they do so in articles about him (fomites); germs may be carried in clothes and other articles, or in ships, especially in the bilge-water. These germs are deprived of activity by a freezing temperature, but may be awakened by a heat of 90° Fahr.; they are destroyed at 250° Fahr.

It has been maintained that yellow fever is only a variety of malarial fever, but the evidence seems to be very strongly against this view.

The National Board of Health (United States, 1879) reaches conclusions about as follows:

The essential cause of yellow fever is a "germ" which is capable of growth and propagation outside of the human body, and which flourishes especially in decaying organic matter. Disinfection must attack the germ and that on which it grows. Disinfection is a poor substitute for cleanliness; it is best done by sulphate of iron, carbolic acid, fresh quicklime, fresh charcoal-powder, chlorides of zinc and aluminium, and permanganate of potash. The disinfectant must be brought into actual contact with the germ; when the germ is dry, it must be moistened or else subjected to a dry heat of 250° Fahr. While the infected articles are dry, move them as little as possible; moisten them with boiling water or some disinfectant solution. The Board advises moist cleansing, followed by fumes of sulphur, using about 18 ounces of sulphur to 1,000 cubic feet of the space to be disinfected. The removal of an unpleasant odor is no proof of disinfection. Textile fabrics which have been exposed to yellow-fever infection should be placed in boiling water or in an oven; rooms and houses which have been occupied by yellow-fever patients should be disinfected as soon as possible. Lastly, they suggest that "every suspicious case of sickness should be at once isolated, and every possible precaution taken to prevent infection, by providing attendants who have had the disease, and thorough disinfection of all discharges from the sick. If the disease proves to be yellow fever, all articles of clothing and bedding used about the sick should be burned, the house should be vacated, and every room tightly closed and fumigated with burning sulphur."

Yellow fever only occurs when the temperature is high; in the northern part of the United States most of the epidemics have begun in July and August. A frost suddenly checks the spread of the disease.

Susceptibility to this affection is greatly influenced by race and acclimation; in many epidemics negroes escape; acclimation may

cause some change in the tissues that impedes absorption of the germs. It is said that acclimated persons are not troubled by mosquitoes, which are usually very plentiful in yellow-fever regions.

Anatomical Appearances.—Rigor mortis is early and decided. The skin is more or less jaundiced. The heart is often pale and relaxed; its muscles may have undergone fatty degeneration. The greatest changes are seen in the abdomen; there is acute catarrh of the mucous membrane of the alimentary canal; rarely there may be ulcers in the stomach, and there is usually more or less dark blood (black vomit is blood mixed with the fluids of the stomach). The liver is but little changed in size, but is of uneven yellow color (more marked in the left lobe), and its cells are filled with fat; it looks like the fatty liver of drunkards. The spleen is not especially changed. The cortical substance of the kidneys is often swollen, and there are signs of inflammation in all parts of them.

Symptoms and Course.—The period of incubation is not settled, but probably is from a few hours to three days. The first symptoms are chilliness alternating with fever, or perhaps a severe chill, great restlessness, red face and eyes, headache ("the most characteristic symptom of the disease is *the peculiar pain in the forehead and eyeballs, with the drunken appearance of the eye*"), pain in the joints, fever (102° Fahr. or more), frequent respiration, and frequent pulse. Severe cases sometimes have a cadaveric odor early in the disease. The tongue is coated and swollen, the pharynx reddened; the gums swell and bleed readily. The epigastrium is very sensitive, and there is usually vomiting. The kidneys are affected, and albuminuria is common; it is said that if the albumen disappears gradually, it is a favorable sign, while its continuance or increase is ominous. The symptoms increase till the second or third day, the temperature rising to about 105°. Then there may be jaundice of the skin and conjunctiva; the urine contains bile; the fæces retain their color, showing that the jaundice is hæmatogenous. There is frequent hæmorrhage from the nose, or more rarely from the stomach; the latter is of evil import.

After increasing in severity for about two or three days, the symptoms subside; the temperature may fall to normal in twelve hours, but does not usually go below 100°; the patient may feel so much better as to think he is well; but the nausea and sensitiveness of the epigastrium remain. This stage of remission may last one or

two days, or it may be the commencement of convalescence. In the third stage the symptoms again grow worse, the temperature rising to 104°; the patient is usually apathetic, though he may completely retain consciousness. Now come the jaundice and hæmorrhages from the alimentary canal; that from the stomach constitutes the "black vomit" which has given one name to the disease. While this "vomit is thick and pasty, being raised in small quantities, and thrown up mixed with natural mucus, the physician does not despair of his patient. The thin black fluid with the coffee-ground sediment is always, in Mobile, a fatal symptom." If the disease continues, the kidney trouble may grow worse, and entire suppression of urine occur. If this third stage is severe, it mostly terminates in death ("from syncope, uræmia, apoplexy, or asphyxia"); but sometimes the temperature falls suddenly, the patient sweats freely, and the severe symptoms all subside. Convalescence is slow, and for a long time the stomach remains weak. In severe cases the strength of the patient may not be "reëstablished sooner than from *ten to twenty days after the cessation of the febrile symptoms.*" "The average duration of fatal cases is less than a week."

The Treatment of yellow fever, as of other epidemic diseases, has varied greatly with different epidemics. Bleeding, salivation, purging, sweating, etc., have each had their advocates; but at present the most favored plans may be considered—first, that of administering at the start a large dose (gr. xx each) of calomel and quinine, followed after a few hours by a purge, and subsequently to treat symptoms; and, secondly, the treatment by laxatives, such as cream-of-tartar and tamarind-water, followed by tonics, and, if the disease goes on, by active stimulants. It is considered very desirable to keep the patients well covered in bed, and in moderate perspiration; the latter may be aided by hot foot-baths under the bed-clothes and by warm aromatic teas. Possibly frequent small doses of jaborandi or its active principle, pilocarpin, might prove beneficial.]

CHAPTER XIV.

SUDOR ANGLICUS.—SUETTE MILIAIRE.—SWEATING-SICKNESS.—FRIESEL FIEBER.

A large number of authorities, especially the Germans, deny the existence of sudor anglicus as a peculiar disease. Thus *Hebra* ignores the well-known fact that the prevalence of this malady is restricted within extremely narrow geographical limits, and that, between the various epidemics of it which have arisen long intervals have elapsed; so, having never met with a case of it himself, he concludes that there is no such disorder. His remarks that there is no febrile affection in which fever-vesicles may not appear, and that the invasion and course of malaria are never accompanied by symptoms which accord with those of sudor anglicus, prove nothing, save that the exanthema is not pathognomonic of this disease, and by no means disprove the fact attested to by many trustworthy authorities, that, besides typhus, acute articular rheumatism, puerperal fever, and many other febrile complaints, there also exists a peculiar sickness characterized by sweating and a miliary eruption more profuse than is often observed in any other disease.

That *suette miliaire*, or the *sweating-sickness*, should be named after one of its prominent symptoms, is quite consistent with the ordinary practice in the case of a disorder not referable to some simple pathological state of a special organ.

Etiology.—Sudor anglicus, beyond a doubt, is an infectious disease. Its exclusively epidemic appearance, its independence of the action of the weather, and other assignable antihygienic influences, as well as the results of the few autopsies which have been made, sufficiently warrant our classing it with typhus, the acute exanthemata, and other disorders which we believe to proceed from infection of the organism by a specific and probably organic poison, and which diseases are certainly more numerous and varied than they are said to be in the schools. Whether the specific poison of the sweating sickness be reproduced in the person of the patient, and thence be transmitted to others; or, in other words, whether it be a contagious disease, is doubtful. All inoculations with the contents of the miliary vesicles, hitherto made, seem to have afforded negative results. This fact, as well as its narrow territorial confines, make it seem more probable that sudor anglicus is not contagious, but that it proceeds from a

miasma, that is, a poison which originates without the body, and which is not reproduced within it.

Regarding the geographical range of sudor anglicus, and the influence of the seasons, and other causative agents upon its epidemic occurrence, *Hirsch*, who has made it the subject of a most thorough historical, geographical, as well as pathological study, speaks as follows: "But few diseases have so limited a geographical range. Its home is in France, southwest Germany, and Italy, while in the Netherlands, middle and northern Germany, and latterly in Spain, it has only been met with in occasional epidemics. In other parts of Europe, and the continents of either hemisphere, it is quite unknown." Of the recorded epidemics, five-sixths broke out in the spring or summer. In autumn they were rare, in winter somewhat more frequent, but never very extensive. The appearance of sudor anglicus does not seem to depend upon any particular atmospheric conditions, and arises as often in mild spring weather as during hot summer. Most frequently epidemics broke out during weather characterized by a moderate temperature and remarkably moist atmosphere. Swampy ground seemed less favorable to its development than a dry, sterile soil. In contrast with many other infectious disorders, sudor anglicus is less frequently seen in large, densely-populated cities, than in small rural communities, country market-towns, and other similar localities. Of the older physicians, *Lancisi*, and more recently *Schönlein*, accuse the water in which hemp has been steeped, of favoring the development of the sweating-sickness.

With regard to the conditions in life which, during an epidemic of this disorder, have been found to predispose to it, it has everywhere been observed that vigorous persons of middle age are peculiarly liable to be attacked, and that women, especially those who are pregnant, or in child-bed, or suckling their children, are more prone to it than men are. In other respects the mode of life seems to have no effect upon the predisposition to the sweating fever. The poor, who usually afford the greatest number of victims to epidemics, do not suffer from sudor anglicus more than the rich.

Anatomical Appearances.—Nearly all authorities speak of the early appearance and rapid progress of putrefaction of the corpse. There are no fibrinous clots in the blood, which is thin and dark of color. The cerebral veins and sinuses are full of blood, and the serum in the ventricles is sometimes increased in quantity. The mucous membrane of the air-passages appears reddened. Somewhat rarely pneumonic infiltration is found in the lungs. The assertion that vesicles similar to those of the skin are found upon the mucous membrane of the intestine as yet lacks confirmation. The spleen is swollen and

soft; the liver filled with blood; the general appearance corresponds with that usually revealed upon the autopsy of an exanthematous patient.

Symptoms and Course.—In a few instances the disease is preceded by languor, headache, soreness of the limbs, loss of appetite, indigestion, and other premonitory but non-characteristic symptoms.

Much more commonly the disorder breaks out suddenly, without any precursory signs whatever. The patient goes to bed feeling perfectly well, and awakes in the night bathed in sweat, the flow of which in a few cases is preceded by a slight chill. He complains of a painful sense of constriction about the precordium, and of an indescribable terror and restlessness. The countenance is reddened, the skin hot, pulse frequent, and urine scanty and concentrated; strange to say, thirst is not always much augmented. Most patients further complain of headache, of a feeling of stiffness and tension about the nucha; many also suffer from mental confusion, dizziness, roaring in the ears, palpitation of the heart, and painful spasms of the extremities, like those of cholera. These are accompanied by a more characteristic symptom, a sense of numbness and pricking of the skin, particularly in the fingers and over the regions where the eruption afterward breaks out most profusely. The sweating is so copious as to saturate the clothing and bedding, and sometimes even the mattress. A peculiar odor has been ascribed to this perspiration by some, who compare it to the smell of rotten straw or musty vinegar; but more probably the odor proceeds from decomposition of the sweat which soaks the bedding.

At the end of about three or four days, the eruption appears, preceded by an aggravation of all the above symptoms, but particularly by an increase of the perspiration, and the prickling of the skin. Scattered here and there among the spots of the eruption, solitary sudamina appear—limpid vesicles filled with sweat, beneath which the skin seems so normal that one might mistake them for drops of water.* The greater part of the eruption, however, is of the miliary form and should rank with the eczemas, since the elevation of the cuticle is not the result of mere perspiration, but of an inflammatory effusion. The vesicles at first are tolerably transparent, but soon become pearly and turbid; and according as they are or are not surrounded by an intensely reddened areola they are described as *miliaria rubra* or *miliaria alba*. Sometimes the effusion accompanying the hyperæmia of the sudoriparous glands in miliaria rubra is so slight that there are apparently no distinct vesicles, but merely solid nodules

* To guard against misunderstanding, the term miliaria crystallina should be disused.

(*eczema papulatum*). The eruption then bears a great resemblance to measles. When the effusion is very profuse beneath the cuticle the vesicles become so large as to suggest a varicella.—The first appearance of the exanthema is upon the sides of the neck and the upper part and front of the chest. Thence it spreads to the belly, back, arms, and legs; it rarely attacks the face or hairy scalp. The eruption breaks out either all at once, the whole body becoming covered with vesicles within a few hours; or else it comes out in crops, one part of the body after another being assailed. The latter is a very common form of the eruption. The fresh outcroppings of the rash are always preceded by an aggravation of the other symptoms, especially of the sweating and pricking of the skin.

Sometimes there is no eruption at all. Such instances are not to be regarded as analogous to the rare cases of scarlatina without exanthema and of abdominal typhus without intestinal lesion. Here the rash seems to depend upon mere sweating; and the latter is the real pathognomonic sign of sudor anglicus. Whether or not there be also a miliary eruption depends a good deal upon the degree of susceptibility of the skin of the individual, just as in cases of sun-burn or of mercurial inunction, it depends in part upon the activity of the irritant and in part upon the sensitiveness of the skin whether or not an eczema solare or eczema mercuriale shall ensue.

When the disease takes a favorable type, the fever and other symptoms assume a remittent and sometimes even an intermittent form, and about the sixth to the tenth day the sweating begins to abate, and no more fresh crops of the vesicles arise. The restlessness also ceases, as well as the headache, prickling, and loss of appetite; the fever subsides, sleep is tranquil, urine copious, the vesicles dry up, and convalescence is established; during which the macerated and loosened cuticle exfoliates, sometimes in large sheets, sometimes in bran-like scales.

Occasionally a relapse interrupts convalescence, and runs a course like that of the original seizure. The malady may thus drag on for weeks, and greatly exhaust the patient, rendering his recovery very slow.

While in many epidemics the favorable type is so much the rule, that the majority or even all patients recover, yet there are others in which the sudor anglicus, without especial complication, makes many victims. It may terminate fatally at any period, death taking place suddenly and without warning, either with symptoms of excessive dyspnœa, or else of cerebral and cardiac palsy. Sometimes the end is preceded by a cessation of the sweating and a fading of the exan-

thema. This has been accounted for upon the obscure hypothesis of a metastasis of the disease to internal organs.

The most common complications of sweating-sickness observed in the more accurately described epidemics are angina and a diphtheritic stomatitis, generally termed aphthæ. More rarely it is complicated with bronchitis and pneumonia. In a few of the epidemics dysentery was observed instead of the usually obstinate constipation. When of very protracted duration, or unusual intensity, then, like other tedious and very severe infectious disorders, it develops a hæmorrhagic diathesis with abundant epistaxis and the appearance of petechia, and in women by abnormal bleedings from the genitals.

Unfortunately, no accurate measurements of the bodily temperature during sudor anglicus have as yet been taken. This circumstance greatly embarrasses our estimate of the physiological value of the symptoms. How, for instance, is the peculiar restlessness to be accounted for? Is it due to inspissation of the blood through enormous sweating, and upon consequent impediment to its circulation through the capillaries of the lungs, as in cholera, with which sudor anglicus has been compared?—Or, is the calorification so much heightened during the fever as to occasion a surcharge of carbonic acid which cannot be neutralized by respiration? Indeed, we are forced to the inference (owing to the considerable cooling which the skin must suffer through evaporation of the profuse perspiration) that the production of heat is much more active in sudor anglicus than in infectious diseases in which the skin remains dry, always supposing the temperature of the diseases to be equal. Cooling of the body by evaporation of the sweat may protect the patient from the perils arising from too high a temperature of the blood, but not from its overcharge with carbonic acid—one of the necessary results of excessive calorification; at all events, this would account for the apnœa of sweating-sickness. Again, does the supposed danger of checking the sweating depend upon the fact that, without the cooling effect of rapid evaporation of the sweat, the temperature will reach a point at which the brain and heart are palsied? Such questions must be deferred until our knowledge of the temperature in sudor anglicus shall be made as exact as that of typhus and other disorders.

Treatment.—Until we shall have more thoroughly investigated the conditions upon which the sweating-sickness depends, prophylactic measures against its invasion and extension are out of the question.

At the outset of the attack an emetic is urgently recommended by many. From all reports, however, it is by no means clear that the disease has ever been cut short by this means; and we shall

therefore do well to confine its exhibition to cases which commence with well-marked gastric disturbance. Its administration, however, at the beginning of a sudor anglicus, during which the bowels are generally confined throughout, is not so hazardous a step as it would be at the commencement of a typhoid fever, when an emetic might seriously aggravate the diarrhœa which always sets in.

The various drugs which have been deemed specifics in certain epidemics have invariably failed in others, and quinine alone seems to have maintained its reputation as an antipyretic. It may be given in doses of twelve or fifteen grains daily, not only when the type of the malady is decidedly remittent or intermittent, but even when the type is continued and the temperature exceeds a certain height. The fact that the danger of the sweating-fever is greatly dependent upon the height of the temperature has already forced itself upon the attention of the best observers, even without the use of the thermometer. When there is no occasion to use quinine, it is best to prescribe acids, especially muriatic or phosphoric acids properly diluted.

An important step in the treatment of sudor anglicus dates from the time when the practice ceased of keeping the patients immoderately warm, for fear that the sickness should "strike in." If my opinion be correct, namely, that the production of heat is intensely active, but that the danger of an overheating of the body is averted by evaporation of the sweat, then it is plain that it would be dangerous to envelop the patient in thick blankets, beneath which the air is so saturated with moisture as to impede further evaporation. Light clothing and free ventilation are imperatively indicated. At the same time the patient must not be set in a draught, lest he take cold, the danger from which is greater in sudor anglicus than in kindred affections, owing to the profuseness of the perspiration.

CHAPTER XV.

CHOLERA ASIATICA.

Etiology.—It is possible, and even probable that, where cholera is endemic, it results from *miasm*. Whether this miasm, the cholera germ, develop in India, on diseased rice or not, and whether or not the disease rage in India, more especially in years when circumstances particularly favor the increase of these vegetable germs, among us, it is not indigenous; but all the cholera epidemics occurring among us are due to the exotic parasite being brought to us by cholera patients, and finding for a time a suitable soil and favorable circum-

stances for increasing; with us cholera is never miasmatic. (I shall not dwell on the question whether *Hallier* and *Klobb*, who have found numerous fungi in the dejections of cholera patients, have actually discovered the cholera germ, for I do not feel competent to decide it.) Cholera is not *contagious* in the strict sense of the word, for a healthy person never takes cholera from contact with a patient having that disease. (My experience in the first cholera epidemic that I saw, where I wrapped many naked patients in blankets, and often held them in my arms for some time, made me a decided anti-contagionist, and I took this ground in my first publication, some twenty years since.) But, as the word contagion is universally used in the sense, that diseases which are transferred from sick to healthy persons are contagious, and as this is very decidedly the case in cholera, we must class cholera among the contagious diseases. The vehicle of contagion is not, as in the acute exanthemata and exanthematic typhus, the exhalation from the skin and lungs, but the dejections of cholera patients. It has been certainly proved that fatal cholera epidemics have broken out in places previously free from it, because some traveller, having cholera germs in his intestines, has used a privy, or that the dejections of a cholera patient have been emptied into a privy, frequented by other persons. In 1848, a transport of recruits from Stettin, where cholera was raging, came to Magdeburg; two of these recruits sickened of cholera the night of their arrival. They were immediately taken from their quarters to the military hospital, which was some distance off, without coming in contact with the inhabitants. A few days after, the cholera broke out in the house, and in the street where they had passed the night. The fatal epidemic did not spread to the rest of the city for some weeks. In large, thickly-populated cities, where there are numerous cases of the disease, the extension of an epidemic is more difficult to follow than it is in small, thinly-settled towns where the number of cases is limited. A small epidemic in Greifswald gave me an excellent opportunity for observing the spread of cholera. In almost every case I could find that the patient had used the privy of some house containing cholera patients, or one whose cesspool communicated with that of the privy of affected houses, or that they had used a privy in common with persons from these houses who had diarrhœa. Since it has been known that cholera is only transferred to healthy persons through the dejections of cholera patients, a series of previously enigmatical and apparently contradictory observations, concerning the spread of the disease, have been satisfactorily explained. We may readily understand that cholera should spread from place to place more rapidly than it formerly did, since in these days of railroads and steamboats people travel more quickly than they used to

do. It is no longer surprising that cholera should follow the routes of travel, that it should spread sometimes with the wind, sometimes against it; sometimes from west to east, again from east to west. The long leaps that cholera epidemics often make are simply due to the fact that travelling cholera patients only infect those places where they leave their dejections, while all intervening places escape. If the cholera germ were contained in the dejections of those patients only who suffered from the severest form of the disease (cholera asphyxia), as they cannot travel, long springs of cholera epidemics could only occur when persons infected with cholera poison travelled during the period of incubation, and the disease did not fully develop in them till they had reached some place distant from their previous residence. But besides such cases, among which are the cases in Magdeburg above mentioned, numerous examples show that a person suffering from simple choleraic diarrhœa, and who does not feel very sick at the time, nor even become so later, contains the germs of cholera, so that he may infect a privy, and thus start an epidemic.

Against the view that cholera was spread by the evacuations of the patients, it has been urged among other things that in some cases persons who had swallowed the dejections of cholera patients escaped the disease, and that attempts to give animals cholera, by introducing into their bodies the contents of the intestines of patients who had died of cholera, or the evacuations of cholera patients, had generally failed. These facts cannot be denied. To make them agree with those above mentioned, it has been suggested that the recent dejections of cholera patients do not contain the cholera germ in the stage of development necessary to infection, nor in the necessary amount, but that the dejections only become dangerous when the germs of the disease are placed under circumstances favorable to their development and increase, by admixture with decomposing animal substances. This hypothesis is very plausible, and has been generally accepted, as it is supported by numerous facts; for, while, according to the observations of *Thiersch*, recent cholera dejections were not dangerous for animals, feeding them with old dejections of the same sort induced cholera. Experience has taught that persons who wash the clothes of cholera patients, after they have lain for some time, and persons who change the bed-clothes a few days after the death of the patient, are more apt to be infected than those who place the bed-pan under the patient, or replace the wet sheets by dry ones. It is most dangerous for the persons in a house, if the evacuations of a cholera patient are emptied into a privy filled with excrement, into a cess-pool, or thrown on a dung-hill. At such places the cholera germ seems to find circumstances most favorable to its development and increase.

When cholera has been introduced into a place, it sometimes happens that only those persons are attacked who live in the same house, or use the same privy. It has even been observed that, in certain cities where cholera has been repeatedly introduced, it has always been limited to these house epidemics. But, in other cases, the disease spreads from the house where it was introduced, to neighboring streets, large portions of the city, or even over the whole vicinity. This may either happen every time the cholera visits the place, or only in certain epidemics, while in others this extension does not occur. We are indebted to *Pettenkofer* for the discovery that porosity of the soil, enabling the contents of the privies and cess-pools containing the cholera germs to freely permeate and soak the ground for some distance around with this dangerous mixture, favors the rapid extension of the disease, while the opposite quality to some extent protects from such an extension. We are also indebted to *Pettenkofer* for the discovery that the occasional predisposition of a place to an extension of cholera depends on the excrements containing the germs, and permeating the soil, being exposed to circumstances favorable to decomposition. As we have already shown, when speaking of the etiology of typhus, moisture of the soil plays a very important part among these circumstances, but it is not the only one. There is no doubt that the conditions for decomposition may be peculiarly favorable when a very moist soil suddenly dries to a certain extent; and it cannot be denied that the sudden fall of the water of the soil may prove very dangerous for the spread of cholera. The opposing facts, which have also been proved, show that the number of cholera cases may increase independently of the sudden fall of the water in the soil, and that it is one-sided to regard the fall of the water as the sole cause for favoring decomposition of the excretory matters, mingled with cholera germs, which have soaked through the soil.

The cholera poison is rarely taken into the system by drinking water containing it. As a rule, it undoubtedly enters the nose and mouth with the air, and is swallowed with the saliva. Using infected privies is so dangerous, because they are the favorite lurking-places for cholera germs, and the gases arising always contain dust-like particles. The poison passes from the privy into the atmosphere of the house, and we must agree with *Biermer* in considering the dwellings as more liable than the inhabitants to infect. Next to the soaking of the infecting substance through the soil, the disease appears to spread from one house to another, chiefly through the gutters and drains.

The susceptibility to cholera poison is very extensive. No age, sex, or constitution, escapes it. When the cholera poison spreads over the whole city, almost every one suffers from it; even those not af-

fected with the severer forms have some troubles, apparently depending on the weaker action of the poison. Certain influences appear to increase the predisposition to the severer forms of the disease, or to diminish the resisting power of the organism to the action of the poison. Chief among these are errors of diet, emetics, and laxatives, catching cold, and other debilitating influences. It is true, foolish people seek to excuse their excesses at the time of cholera epidemics by saying that the mode of living can have no effect in inducing cholera, because persons who lead the most proper lives are attacked by and die of the severest forms of the disease. Even if this reasoning was meant in earnest, it does not require refutation. Whoever is exposed to a poison, whose action kills many persons, while others recover from it, is foolish to subject himself to injurious influences which lessen his chances of recovery, even if the avoidance of these injurious influences gives no guaranty for a favorable termination. The number of cholera patients taken into the Paris hospitals is said to be one-eighth greater on Monday than any other day. In the Magdeburg epidemics, the commencement of a fair, which gave opportunity for excesses of all sorts, has repeatedly been observed to have a very unfavorable influence on the number of cases and deaths.

For the numerous important historical and geographical data that have been collected concerning cholera, since 1830, when it first appeared in Europe, I must refer to special works where the principal epidemics are fully described; for imperfect extracts from them would be unsatisfactory.

Anatomical Appearances.—Bodies of patients who have died of cholera remain warm a long time; a *post-mortem* increase of the bodily temperature has sometimes been observed. A second peculiarity of these bodies is the occasional contraction of certain muscles after death, by which the extremities, especially the fingers, are moved, and change the position they had just after death. The movements of the fingers that I have actually seen, and the changed position in which I have found the bodies a few hours after leaving them, have always made a very disagreeable impression on me.

If death has occurred at the height of the disease, the appearance of the body is characteristic. We usually find it in a position to which the clinched hands, variously bent limbs, and swollen muscles, give a peculiarly threatening appearance ("fighting attitude"). The rigor mortis is hard to overcome. The face is often so distorted as to be hardly recognizable. The eyes are deep in the orbit, and are surrounded by wide, blue rings; the eyelids are half closed; the uncovered portions of the eyeballs are dry as parchment, the nose is pointed and projects far beyond the sunken cheeks. The lips are bluish,

sometimes deep blue. The other parts of the body also have a more or less marked cyanotic appearance. This is most apparent in the terminal phalanges of the fingers and toes. The skin of the fingers is often shrunken, and wrinkled like that on the fingers of a washerwoman who has had her hands in the suds all day. On opening the bodies we remark the hardness and dryness of the subcutaneous connective tissue, and the dark-red color of the muscles. The blood is thick, of the color of huckleberry-juice, and contains a small amount of soft, black clots; it is collected in the right heart and veins, while the arteries and left heart are often perfectly empty. The cerebral sinuses and veins of the meninges are distended with dark blood, while the cerebral substance is dry and hard. The pericardium does not contain a trace of serum, its inner surface feels pasty, and is often covered with ecchymoses; the muscular substance of the heart is contracted, hard, and of a dirty-red color. The surfaces of the pleura, like those of the pericardium and other serous membranes, are covered with an adhesive layer. They also often contain small ecchymoses. On opening the thorax, the lungs collapse very quickly and completely, apparently because the empty, dry bronchi offer no opposition to the escape of air from the alveoli. On cutting into the lungs, we find them remarkably dry, and free from hypostasis and œdema. The relaxed, baggy small intestine has a peculiar rosy appearance, even before it is opened, while the large intestine preserves its natural color. On opening the intestines, quantities of a colorless or faintly-colored fluid, containing white flocculi, often escape; this liquid exactly resembles the "rice-water discharges" of cholera patients, which we shall more minutely describe. I have found the greatest amounts of transudation in the intestines, in cases of so-called cholera sicca. The mucous membrane of the small intestine is finely injected, especially near the valve, and growing less so as we pass upward. The vascularity is occasionally accompanied by greater or less escape of blood into the tissue, and on the free surface of the mucous membrane. Then the mucous coat shows numerous, often extensive, ecchymoses, and the contents of the intestines appear reddened from the admixture of blood. Sometimes the small intestine is pale, and neither vascularity nor ecchymoses can be found; but since we find the intestines filled with watery transudations even in such cases, and as these transudations come from distended, not from empty vessels, it follows that the pale hue of the mucous membrane is to be regarded as a *post-mortem* appearance. It is a matter of daily experience for visible mucous membranes that have been very hyperæmic, and secreted abundantly during life, to become pale after death. The mucous membrane and whole intestinal wall are swollen and relaxed from œdematous infiltration. As a rule, also, the

solitary and *Peyer's* glands are swollen and distended. The individual follicles may attain the size of a hemp-seed. In consequence of this swelling of the intestinal glands, the inner surface of the bowel looks as if sprinkled with isolated and conglomerated granulations. Occasionally certain follicles of the patches burst, and then the surface has a sieve or net like appearance (*plaques à surface réticulée*). The most important appearance in the intestine is the great loss of epithelium The intestinal villi are stripped of their protecting envelope; occasionally, at certain spots, the epithelial covering is only elevated by a serous effusion, and is still loosely adherent to the villi; but in most places it is already detached, and lies on the intestinal wall as shreds of mucus, or forms the whitish flocculi already spoken of as floating in the transudation. The comparison of the intestine in cholera to a portion of the skin from which the epidermis has been removed by a blister, or by boiling water, is very correct; and, if it be remembered that the denuded portion of intestine is quite extensive, it will be difficult to understand why some observers speak of a "disproportion" between the anatomical changes in the intestinal canal and the severe symptoms observed during life. The large intestine does not show any constant changes, and there is but little alteration in the jejunum. The gastric mucous membrane is more or less reddened by hyperæmia and ecchymosis; its tissue is swollen and relaxed from serous infiltration. The liver is of normal consistence and pale; on being incised, only a small amount of thick, huckleberry-colored blood flows out slowly from the large vessels. The gall-bladder is almost always distended with thin, brownish or greenish bile. The spleen has no constant change. In the first stages of cholera the kidneys are apparently normal, except an excessive venous hyperæmia; in other cases, even at this time, certain places, especially in the pyramids, are whitish; and at these points, on microscopical examination, we find the uriniferous tubules filled with cloudy, swollen epithelium and fibrous exudation. The mucous membrane of the urinary passages is covered with mucous and epithelial masses, the bladder is contracted, and almost always entirely empty. Hence we see, when death occurs at the height of the disease, *the characteristic changes consist chiefly in extensive catarrh of the intestines, accompanied by detachment of the epithelium and copious transudation, in decided thickening of the blood and excessive venous hyperæmia of the kidney.*

When death occurs during the stage of reaction, or so-called cholera typhoid, the anatomical appearances differ in some respects from the above. Then the limbs are less constantly contracted, rigor mortis is less marked; the teeth and gums are often covered with a dirty coating, the cyanosis has disappeared or is slight. The subcutaneous con-

nective tissue and muscles are moister; the blood is more fluid and less dark. The cerebral membranes are usually injected; not unfrequently there is a considerable amount of fluid in the meshes of the pia mater and in the lateral ventricles; the cerebral substance itself is moister; the right heart is usually overfilled, the endocardium and lining membrane of the large vessels is greatly infiltrated. In this stage the lungs are no longer dry, but are vascular, and are often the seat of extensive œdema and hypostasis, and not unfrequently also of lobular or lobar pneumonia, or of hæmorrhagic infarctions. The outer surface of the small intestine has lost its rosy tint; the contents are colored with bile. In some cases the epithelium is replaced, and no disturbances of nutrition are discoverable in the mucous membrane; but it is often the seat of a typical diphtheritic inflammation, which changes more or less extensive patches of the mucous membrane into brown, dry sloughs. This secondary diphtheritis occurs not only in the small intestine, but often extends to the large intestine; it may also attack the gall-bladder, vulva, and vagina. The liver and spleen are not always changed, but are usually very hyperæmic. Not unfrequently rupture of the spleen has been found. The kidneys also are vascular, and often show the signs of acute croupous inflammation. The bladder contains more or less urine, which is usually albuminous.

Symptoms and Course.—Almost every one exposed to cholera poison complains of light oppression in the præcordium, rumbling in the bowels, and a feeling of impending diarrhœa. These symptoms of slight indigestion, which unmistakably result from the action of the poison, apparently only increase where the infection is rather intense, or the organism inclined to a more or less severe disease. It has also been attempted to refer the feelings of terror, the fainting-fits, cramps in the legs, and other disturbances of innervation occurring during a cholera epidemic, to the action of cholera poison; and among the laity the idea has become so firmly implanted, that the fear of cholera is very dangerous, or is even the commencement of the disease, that during a cholera season there are plenty of persons who dread having a fear of the cholera (!). I regard this belief as groundless, and think that the above symptoms are solely the result of the psychical impressions induced in excitable persons, by the terrible disease, the accounts of sickness, the numerous and unexpected cases of death. The same, or very similar symptoms are experienced by the inhabitants of a bombarded town; and, although timid persons show no immunity to cholera, they are not more frequently attacked than unterrified persons are. According to my observation, cholera attacks never begin with a feeling of terror, fainting, cramps in the legs, etc., although it often

happens that these symptoms urge the patient to seek medical aid. If we examine such cases carefully, we always find that they have been preceded by diarrhœa, to which the patient paid no attention.

Some observers estimate the period of incubation at from one to three days; others say from eight to fourteen. We rarely have the opportunity of exactly determining the time between the action of the poison and the outbreak of the disease. In a few cases that I observed in Greifswald, in 1859, as well as in a number observed by Dr. *Grüttner*, then assistant physician in the Medical Polyclinic, in a small town on the Mecklenburg boundary, where the limits of the infection could be pretty accurately determined, the period of incubation was certainly not less than thirty-six hours, and not more than three days.

The mildest form of cholera is a simple diarrhœa, which is not accompanied by colicky pains or tenesmus, and causes no constitutional or other disturbance, except a moderate degree of depression and relaxation. The evacuations follow each other more or less closely; they are very copious and watery, but have neither lost their odor nor color. These cases do not appear on the official lists as cholera; but, although the police do not consider them as such, science should do so. This is shown: 1. By the large number of cases of diarrhœa occurring in cholera times, although almost all sensible people carefully avoid errors of diet, catching cold, and other sources of injury. 2. The great obstinacy of these diarrhœas, and the slight efficacy of opium against them. 3. The well-known transportation of cholera by persons suffering from these diarrhœas. 4. But especially the numerous transformations of simple "cholera-diarrhœa" into the severest forms of the disease. Many patients, especially of the poorer classes, worried by a diarrhœa that would not give way to domestic remedies, go to the doctor's house for a prescription at noon, and in the evening lie cold, pulseless, and cyanotic, in an almost hopeless state. The investigations concerning cholera, made during the late epidemic, especially in the hospitals, which are otherwise very serviceable and valuable, have made some believers in the false views concerning the significance of the intestinal affection in cholera, which I combated twenty years ago It has again been forgotten that very many cholera patients, who do not seek admission into hospital, have no symptoms but the profuse diarrhœa. I consider it much more important to determine the frequent occurrence of a gradual transformation from simple cholera diarrhœa to cholerine, and to malignant cholera, and to prove the identity of these three forms, than to seek for pathognomonic signs of epidemic cholera.

The transformation from the mildest forms of cholera to the severest

is formed by those cases where violent vomiting accompanies the diarrhœa, where the discharges acquire the well-known "rice-water" appearance, but without the occurrence of the paresis of the heart and thickening of the blood, which constitute the terrible symptoms of cholera asphyxia. This still mild form, or, rather, these comparatively low grades of the disease, which, however, often increase to the highest grades, have been distinguished from cholera diarrhœa on the one hand, and from cholera asphyxia on the other, by the names "erethitic cholera," or "cholerine." The discoloration of the dejections depends chiefly, or entirely, on their excessive dilution by the quantities of fluid transuded into the intestine; hence the more copious the passages, and the more rapidly they succeed each other, the more completely and speedily they lose their brown color and fecal odor. Occasionally all the contents of the intestine are passed at the first evacuation. In such cases, even after the second movement, the dejections consist of an almost colorless and odorless fluid, holding in suspension more or less white flocculi. We must not conclude, from the lack of color in the passages, that the formation or excretion of bile has ceased; for even if the bile be produced in normal amount, and poured into the intestines, it can have no great effect on the color of the large quantity of liquid. Chemical and microscopical examinations of cholera stools have shown that the serum, transuded from the intestinal capillaries, contains little albumen, but plenty of salts, especially of chloride of sodium, and that the white flocculi floating in the serum rarely consist of perfect cylindrical epithelium, but generally of its remains, in the shape of fine, loose nuclei, with coarse and fine granular masses embedded in a mucous basement substance, and of round, nucleated, coarse, or finely-granulated cells (*Bruberger*). Cholera-stools occasionally contain crystals of triple phosphate, remains of food, parasites, vibriones, and fungi. Lastly, the dejections sometimes contain blood-corpuscles; then the fluid is somewhat richer in albumen, which has escaped from the capillaries along with the blood. These characteristics of cholera-stools, which all authors consider pathognomonic, fully explain the symptoms of cholera. We are justified in comparing the effects induced in the bowels by cholera poison, with those produced on the skin by a blister. In both cases the protective covering is removed, and there is an excessive transudation from the capillaries. It only depends on the intensity, and particularly on the extent, of the process, whether symptoms of paralysis of the heart develop, and whether the loss of water from the blood shall prove dangerous. Cases where the heart's action is but little weakened, and where the loss of water from the blood is to some extent replaced, correspond to cholerine. When the characteristic cholera stools begin, the thirst already

caused by the simple cholera diarrhœa is decidedly increased. This torturing symptom hardly requires explanation, as it always follows loss of water from the blood, whether induced in fevers by the increase of insensible perspiration, by sweating, or by increased excretion of urine. In cholerine the loss of water from the blood is greater, and consequently the thirst is more intense than in cholera diarrhœa. The characteristic evacuations, severe thirst, weariness, and depression, are usually accompanied by a very annoying symptom, which it is difficult to explain; there are occasional spasmodic contractions of certain muscles, especially of the calves of the legs, which last from half a minute to a minute, and are very painful. These cramps are not pathognomonic of Asiatic cholera, however, for they also occur in severe attacks of cholera morbus. In favorable cases, the discharges gradually become less frequent and copious; the bile poured into the intestines again suffices to color the passages. Finally, the diarrhœa ceases, and the patient begins to recover; but convalescence is always slow. In other cases the disease relapses after it appeared to be doing well, and then becomes dangerous. Lastly, in still other cases there is no improvement, the cholerine changes to cholera asphyxia.

Cholera asphyxia depends on the highest development of the intestinal affection. At least all its characteristic symptoms may be directly referred to the severe and *extensive* disease of the intestinal mucous membrane and to the copious exudations from the intestinal capillaries. The accounts of persons dying during cholera epidemics with the symptoms of pulselessness, cold skin, cyanosis, etc., who had had neither diarrhœa nor vomiting, and in whose intestines no characteristic changes were found, have become more rare in late epidemics; so that at present almost all experienced physicians deny the occurrence of "cholera sicca," which was generally considered as proved in the first cholera epidemics. But the case is different in regard to the views about the dependence of the other symptoms of asphyctic cholera on the intestinal disease. Many physicians, who consider the latter as constant, do not refer the other symptoms of cholera to it, but think that the intestinal disease in Asiatic cholera has no more effect on the general appearance of the disease than the bowel lesion in abdominal typhus has on the symptoms of that affection. We shall again refer to the erroneousness of this view. In many cases asphyctic cholera develops from a cholera diarrhœa or a cholerine that has existed for several days; but, fully as often, the symptoms to which this form owes its name come on a few hours after the first cholera passage. By this all the contents of the intestines seem to be evacuated; the patients are astonished that the vessel which they used is nearly filled, but do not suspect that they are in great danger and neglect to seek aid for

the simple and painless diarrhœa, while previously they have perhaps worried the physician about every insignificant colicky pain. The first passage is soon followed by a second, this by a third, and so on till a great number have occurred at short intervals. The evacuations are very copious and fluid, and, as they lose their fecal odor and become colorless, they soon acquire the rice-water appearance. Even after the second or third passage, many patients feel very weak and depressed, or they may be so faint as to be unable to move from the close-stool to the bed without assistance; at this time, also, painful contractions of the muscles of the leg usually begin, and a longing for drink, which increases with every passage. The more the patients drink, the sooner the diarrhœa is accompanied by vomiting, by which at first merely the contents of the stomach, but, after a time, large quantities of a pale, yellow liquid, are evacuated. The patient rapidly grows weak, the voice loses its power (vox cholerica), the evacuations are passed involuntarily, the secretion of urine ceases, the painful muscular cramps increase and return more frequently; the torturing thirst cannot be allayed, and these symptoms are accompanied by a feeling of great anxiety and oppression, which, together with the cramps in the legs, forms the most painful symptom of cholera. Meantime the appearance of the patient has become frightful; the eyes are sunken, the nose pointed, the cheeks hollow (facies cholerica); the skin of the hands is wrinkled like that of a washerwoman who has washed all day; if it be picked up in a fold, the fold remains for a time and disappears slowly. The lips, extremities, and genitals, grow blue; the whole surface sometimes assumes a bluish or grayish look. The radial pulse, which becomes smaller after the first passages, frequently cannot be felt an hour after the commencement of the cholera attack. Finally the pulse disappears from the carotids also, the impulse and sounds of the heart become indistinct, and, while the circulation grows more imperfect, while less warm blood reaches the periphery, the temperature becomes corpse-like, particularly at the uncovered parts (stadium algidum). Rarely the patients complain of headache, more frequently of black spots before the eyes, noises in the ears, or dizziness. The mind is not cloudy, but most patients are apathetic; while they complain of pain and oppression they are indifferent to the danger, and answer inattentively and slowly. Reflex excitability is diminished; in severe cases even irritating vapors induce neither coughing nor sneezing; the patients do not wink if the finger be approached to the conjunctiva, and do not wince if we dash water on them. It cannot be wondered at, that in the first cholera epidemics even those physicians who regarded rice-water passages as pathognomonic of cholera, who ordered careful anti-diarrhœic regimen for their patients, and treated every diar-

rhœa energetically, should not have gone a step further, and recognized the intestinal lesion as the starting-point of the other symptoms and as the true source of danger. The rapidity with which the patients changed, the great disturbance of all the functions, the pulselessness, coldness, suppression of urine, vox cholerica, facies cholerica, lack of contractility in the skin, and the fact that many patients were received into the hospital in this state, who had no diarrhœa or vomiting after their reception, and who did not always tell that they had previously had violent evacuations, led to untenable hypotheses. It is true it was acknowledged that cholera poison led to an affection of the intestinal canal; but at the same time it was accused of having a directly pernicious effect on the blood, nervous system, and more or less on all the organs and tissues, the intestinal canal escaping altogether sometimes. Cases where the algid stage develops in a few hours are certainly less suited to explain the dependence of all the symptoms on the intestinal affection than are those where it develops in the course of several days. From the identity of the symptoms that finally result, however, we cannot doubt that the rapid cases are to be explained in the same way as the others. The immediate result of acute intestinal catarrh, of the excessive transudation from the intestinal capillaries, and of the diminished absorption of the fluid drank, is a thickening of the blood, its sudden impoverishment in water and salts. As long as the disease is moderate, it has no particular effect on the circulation and distribution of blood through the body; only the thirst is increased and the secretion of urine diminished. But, as a burn of the second order is free from danger as long as it affects only a limited portion of the surface, while it becomes very dangerous if widely extended, and as we dare not deprive the entire surface of its epidermis by blisters, so an extensive and intense choleraic affection of the intestines induces the severe and threatening symptoms that characterize the algid stage. The heart's action is palsied, the blood, deprived of its water, eagerly takes the fluid from the interstices of all the tissues. Hence the tissues all become dry and diminished in size; the nose becomes pointed, the cheeks hollow, the eyes sink into the orbit, the skin of the fingers shrivels, and, when pinched up, stands in folds. Even pathological collections of fluid, which had previously resisted all treatment, effusions in the pleura, joints, etc., are absorbed. Moist eruptions and ulcers acquire a parchment-like surface. In spite of the patient's drinking constantly, the loss of fluid so far exceeds the supply that he may lose one-fifth of his weight in a few hours. The thickening of the blood explains the drying up of all the secretions of the saliva, tears, sweat and urine, just as well as it does the absorption of the interstitial fluids; the blood actually does not contain the material

for these secretions. In the suppression of urine, however, the stagnation of the circulation also plays an important part. The feebleness of the heart's action, which causes the weakness and indistinctness of the impulse and tones of the heart, the diminution and disappearance of the pulse in the radial arteries, and even in the carotids, appear to depend chiefly on the depressing influence that severe acute disease, especially of the abdominal organs, has on the organic nervous system, and particularly on the nerves of the heart. Immediately after the perforation of an ulcer of the stomach I have often seen loss of pulse, cyanosis and coldness of the extremities, and in one case, already mentioned, perforation of the duodenum, was diagnosed as cholera sicca. Such cases, where there could not be the slightest suspicion of an infection, show that the hypothesis that the cholera-poison poisons the sympathetic, is untenable. On the other hand, it is not improbable that the stagnation of blood in the capillaries of the heart's substance has something to do with the paralysis of that organ. We know that blood can only pass freely through the capillaries when the blood-corpuscles are separated from each other by a sufficient amount of intercellular fluid. Hence a loss of water from the blood, such as occurs in cholera, must hinder or even arrest the circulation in the capillaries; and, if the blood in the capillaries of the heart stagnate, according to all physiological and pathological experience, paresis of the heart is the inevitable result. The cyanosis that occurs in the algid stage of cholera depends on the same cause as that occurring in other diseases, on an abnormal distribution of the blood—the arteries, which receive no blood from the heart, contract and press their contents into the capillaries and veins; but the collection of the blood in these vessels causes very great cyanosis in cholera, because the blood is so concentrated, and hence is relatively rich in colored corpuscles, and because, from the retardation of the circulation, it has become so venous in character, and consequently very dark. If an attempt be made to bleed in the algid stage, as was often done in the first epidemics, a thick, dark stream springs from the swollen vein, but no more blood follows the first spirt, the stream soon ceases, and then it is difficult to bring out even a few drops by pressure and rubbing. As the circulation is reëstablished, the cyanosis rapidly disappears, although the blood still remains of a dark, huckleberry color. As early as 1848, in my pamphlet, "Die Symptomatische Behandlung der Cholera," I showed that the cyanosis and asphyxia did not depend exclusively on the thickening of the blood, but was mostly due to the paralyzing influence of the extensive intestinal disease on the sympathetic nerve. The correctness of this view is supported by the frequently rapid disappearance of the cyanosis, etc., by its ceasing before the thickening

of the blood could possibly be remedied by the absorption of liquid. The stagnation of blood in the capillaries of the lungs, induced by the paralysis of the heart and thickening of the blood, explains one symptom, for which I could give no explanation in 1848, that is, the feeling of anxiety and oppression, which is rarely absent in the algid stage. The change of the blood in the pulmonary capillaries is just as necessary for the respiratory act as the change of air in the air-cells; and stagnation of the circulation induces the feeling of want of air and oppression, just as is done by obstructions in the bronchi and alveoli, which impede the entrance and exit of air. The very slight amount of carbonic acid in the air expired by cholera patients shows that, in spite of the extensive movements of the chest and the unimpeded entrance of air to the air-cells, respiration is imperfectly performed. Lastly, the complete arrest of the secretion of urine in the algid stage, and its diminution even in cholerine and cholera diarrhœa, are easily explained. We know that the amount of urine secreted depends chiefly on the amount of lateral pressure in the glomeruli of the Malpighian capsule; we have already explained, that in heart and lung diseases, which induce imperfect filling of the heart and arteries, the secretion of urine is diminished; hence it is not strange that the urine should be suppressed in the algid stage of cholera, where the heart's action is reduced to a minimum, and the pulse cannot be felt even in the large arteries. The low temperature of the periphery of the body appears partly due to the diminished production of warmth, partly to the diminished supply of warm blood to the skin (from the weakened action of the heart).

Cholera asphyxia runs a very acute course. Many patients die in six, twelve, or twenty-four hours. The algid stage rarely lasts longer than two days. The evacuations often cease some time before death, and we must be careful about regarding this as a favorable sign, as it is not due to cessation of the transudation, but to paralysis of the intestinal muscles. On the contrary, patients in whom the evacuations continue for a long time recover more frequently than those in whom they cease suddenly. It would be wrong to decide from this fact that an excessive and long-continued transudation into the intestine had a favorable influence on the course of the disease, or was at least unimportant. It would be much more correct to interpret the fact thus: In cholera, the occurrence of paralysis of the intestines is one of the most unfavorable symptoms, and the continuance of the evacuations shows that the intestines are not yet paralyzed, and so justifies a more favorable prognosis. The death of the patient is a gradual "going out;" the rattling in the throat especially, which takes place shortly before death in most diseases, is absent. In favorable cases the pas-

sages become fewer and less copious, and the liquids taken into the stomach are no longer vomited. The first signs of improvement, which always introduce the change in the disease, are soon followed by symptoms which show that part of the liquid taken is absorbed, and that the blood is consequently thinned. The capillary circulation is restored, the pulse returns to the carotids, then to the radial arteries; the cyanosis disappears; the skin resumes its color, and the face loses its distorted appearance; the disease passes from the algid stage to that of reaction. Occasionally, this stage offers no peculiarities, and forms the beginning of convalescence; then, when the asphyctic symptoms have ceased, there are a few more copious passages, with distinctly fecal odor. Even on the second or third day there are pulpy or formed stools, or else constipation. Every thing indicates that the lost epithelium has been regenerated. We may compare these cases to those where a superficial dermatitis from a blister has been entirely removed in a few days by regeneration of the epidermis. When the stage of reaction forms the commencement of convalescence, even the stagnation in the capillaries during the algid stage has not led to any considerable disturbance of nutrition in any organ; only there is exceptionally albumen in the first urine evacuated, on account of the stagnation in the veins that precedes the reëstablishment of the normal circulation. In other cases, where the damage to the intestine is less quickly and completely repaired, the violent evacuations cease during the stage of reaction, but a moderate diarrhœa, with fluid, badly-smelling greenish stools, continues; the pulse remains small; the temperature of the extremities low, and the patients are in great danger of dying from exhaustion on an exacerbation of the intestinal disease. But there is not generally a return of the algid stage with disappearance of the pulse, cyanosis and coldness of the body; the incomplete reaction is more apt to pass into the so-called typhoid stage of cholera, but not unfrequently it ends in protracted convalescence, after the diarrhœa has ceased. Sometimes after the algid stage the pulse not only returns, but becomes unusually full and strong, the previously sunken temperature rises above the normal height, the cheeks turn dark red, the eyes are injected, and the signs of fluxionary hyperæmia to the brain and other organs appear. These violent reactive symptoms are difficult to interpret. They most probably depend, at least partly, on the abnormal quality of the blood, and the consequent impediment to the capillary circulation. The symptoms of violent reaction also pass imperceptibly into those of the typhoid stage, or into convalescence. The opinion, which I advanced from my observations of the first cholera epidemics, that the temperature was only diminished at the periphery while it was elevated within the body, has been

proved to be correct on the whole. From numerous careful observations of the temperature in cholera patients, *Jüterbogk* arrived at the following results: 1. In the algid stage, the head, extremities, etc., are colder than in almost any other disease. 2. In the algid stage, the temperature of the cavities of the body, such as the vagina and rectum, is the highest (that can be measured) in the body, and it should always be taken for measurements. 3. In the algid stage, whether the case be favorable or fatal, the temperature within the body is usually increased, more rarely normal, most rarely diminished, although no cause for this has ever been found in the pathological symptoms during life, or on autopsy. 4. In the algid stage, the temperature of the whole body usually rises with the approach of death, but does not appear to increase afterward. But there are cases where this rise does not take place without our being able to find any reason for this deviation. 5. The commencement of reaction is not accompanied by any elevation of temperature, but the interior of the body usually cools off, while the outer parts warm up. 6. In cases of protracted reaction, the temperature of the whole body generally sinks below the normal. 7. The inflammatory sequelæ generally, if not always, cause a decided elevation of temperature. 8. During perfect convalescence, an abnormal elevation of temperature is often seen, without any pathological cause for it being discoverable.

The general name of "cholera typhoid" has been given to the secondary symptoms that often follow the proper cholera attack. From the fact that these sequelæ follow cholera asphyxia almost exclusively, never simple cholera diarrhœa, rarely cholerine, and do not constantly follow the first form of the disease, we may conclude that they do not directly depend on infection with cholera poison, but are based on the pathological processes during an attack of cholera, particularly of the severest form. As we have already seen, the same state of affairs occurs in typhus, where also the proper symptoms of poisoning are often, but not always, followed by secondary affections due to the typhus. It may be readily seen that the stagnation of the thickened blood in the capillaries, and the consequent interruption of nutrition, if they last for several hours, a day, or more, may have a very injurious effect on the nutritive condition and functions of the organs; and we have already mentioned a series of inflammatory symptoms whose remains were found in the bodies of persons who died after the termination of the actual cholera attack. This view of the origin of the secondary diseases (the cholera typhoid) also agrees with the fact that they most frequently occur when the algid stage has been very marked and protracted. The fact that the secondary inflammations remain more or less latent, and often betray them-

selves only by symptoms of excessive adynamia ("typhous symptoms"), is a peculiarity shown also by other inflammations when they attack debilitated persons. We shall only call attention to the fact that in old, decrepit persons, if physical examination be neglected, the outward resemblance and the subjective symptoms often cause pneumonia to be diagnosed as catarrhal fever, nervous influenza, typhus, etc. According to my experience, acute croupous nephritis, with the retention of urine that it causes, by plugging up the uriniferous tubules, is the most frequent sequel of cholera asphyxia, but is by no means the constant cause of cholera typhoid, as is often asserted. If the secretion of urine remain suppressed after the disappearance of the symptoms of collapse, or if the scanty urine contain quantities of albumen and fibrinous casts for days, if vomiting recommence, and the patients complain of severe headache, and become comatose, or have epileptiform convulsions, we may make a diagnosis of acute croupous nephritis and so-called uræmic intoxication. In such cases the skin has occasionally been found incrusted with crystallized urea. The first or second day after the cessation of the asphyctic symptoms many patients pass a normal or even very large amount of urine; and the albumen, which is at first constant in it, usually disappears after a few days, nevertheless they fall into a state of great apathy and stupor, or muttering delirium, the tongue becomes dry and crusted, the pulse frequent, and often double; the temperature is elevated; the patients slip down toward the foot of the bed, and the disease so exactly resembles a severe typhoid, that there is no doubt the name cholera typhoid was intended for these cases. Besides the above symptoms, there is usually diarrhœa, with fetid evacuations mingled with shreds of epithelium; and, while the patients can scarcely be aroused from their comatose state by loud cries or other irritants, they twitch the face or recover consciousness, and complain of pain, if strong pressure be made on the abdomen. In these cases there is diphtheritic inflammation of the intestines, which often succeeds the catarrhal or proper choleraic enteritis, and which is perhaps induced by the irritation of the denuded intestines by their contents; most patients who fall into this state die of exhaustion. If, instead of diphtheritic inflammation of the intestine, there be a similar affection of the genitals, a pneumonia, pleurisy, or some other inflammatory sequel of cholera, the appearance of the patient does not materially differ from the above description. The typhoid symptoms due to the fever prevail, and the subjective symptoms of the local disease fall into the background, or disappear entirely. Lastly, in some cases, neither during life nor on autopsy can we find any local disease to which to refer the exhausting fever, of which many patients die after the cholera proper has run its course.

Peculiar importance has been attached to the fact that, during the so-called cholera typhoid, a maculated, papular, or erythematous exanthema has been observed; and the "cholera-exanthema" has even been compared to the "typhus-exanthema," and its occurrence regarded as a proof of the similarity or relationship of cholera typhoid and typhus. The exanthema is not, however, so constant a symptom of cholera typhoid as to be pathognomonic, and according to my observation it occurs chiefly in those cases where sinapisms have been applied repeatedly or continuously to the extremities during the algid stage, or where the extremities have been rubbed energetically. The exanthema, which chiefly affects the limbs, often extends to the trunk, hence its occurrence seems to me to be due to continued stagnation of the circulation, and consequent interruption of the nutrition of the skin, and to be favored by precedent irritation of the skin. Moreover, attention has recently been called to the fact that the diagnostic importance of the exanthema of typhoid fever has been overvalued, and that roseola spots and erythema also occur in many other feverish diseases.

Treatment.—We shall not discuss the sanitary police regulations by which we may hope to arrest the progress of cholera epidemics; and shall only call attention to the fact that, in the Mecklenburg epidemic of 1859, it was shown that the quarantining and locking up, which, from the experience of previous epidemics, were declared to be utterly useless, were found to afford full protection when energetically and perseveringly followed out. Since a person, suffering from an apparently simple and harmless diarrhœa, may carry the cholera-poison to a previously healthy place and there induce a fatal epidemic, places that would be protected must cut themselves off from *all communication* with the rest of the world. It would be very satisfactory if the attempts to dry the soil of cities by drainage, and by improving the erection of privies, should have the desired result of lessening the predisposition of the affected places for extensive cholera epidemics. It would also lead us too far, were we to treat fully of the police regulations that physicians, in places where the cholera has appeared, must require of the proper authorities, and here we can only make certain suggestions. Since the privies, cess-pools, dirty gutters, etc., favor the development of the cholera-poison, they should be energetically cleaned and disinfected. Stools of cholera patients should never be thrown into the common privy. One of my pupils, Dr. *Reich*, while still a student at Greifswald, was cholera physician at Tribsees, a small town on the Mecklenburg border, where he succeeded in urging the police to empty all the privies and have a certain quantity of solution of sulphate of iron poured into them. Large tanks, filled with this fluid, were placed before each house to facilitate this procedure,

which was strictly enforced on the inhabitants. But it has not been proved, on the contrary, it is much doubted by competent authority, whether sulphate of iron, which so well removes the unpleasant smell from privies, also interferes with the development and increase of the cholera germs. The epidemological section of the Berlin Academy of Medicine recommend the disinfection of body and bed linen by boiling in water; for the disinfection of privies, chloride of lime (ten parts, in solution, to one hundred of fæces); for bed-pans, night-stools, etc., they recommend a mixture of two parts of permanganate of soda, forty-five parts of sulphate of iron, and fifty-three parts of water (ten parts to one hundred of fæces, or a wineglassful for each person); for the disinfection of dwellings where cholera patients have been sick, chlorine gas. Physicians should also persuade the proper authorities to have prepared sufficiently large and properly-constructed lazarettos, well supplied with nurses, where patients with suspicious diarrhœa may be separated from those with well-marked cholera; they should urge the supply of healthy nourishment to the poor by soup and eating houses, and that the people may be informed, by temperate and simple publications, of the danger they run by neglecting an ordinary, painless diarrhœa. Lastly, where it is possible, places should be arranged where the inhabitants of houses that have been attacked may find shelter.

The prophylactic rules for physicians to recommend to their own patients, on the outbreak of a cholera epidemic, are as follows: since there is far more danger in a city where cholera is prevalent, and still more in a house where it has broken out, than in other places, it is sensible for persons, who can make a long journey without great inconvenience, to fly from the disease. Such persons should be sure—1, to start on their journey soon enough; 2, to go off as far as possible; 3, not to return till the last trace of the disease has disappeared. We should strictly forbid those who must remain from using a strange privy. It is remarkable that this important and certainly not superfluous advice is not given in the cholera regulations published by *Griesinger*, *Pettenkofer*, and *Wunderlich*. If I were writing regulations for the cholera, before treating of disinfection of privies, I should urge not to rely on it, and not to visit even a carefully-disinfected privy that is used by strangers. There are many heads of families who would not object to buying a night-stool for themselves and family during a cholera epidemic. We should also let our patients be careful about their diet; i. e., avoid all food that is difficult of digestion, and all articles of diet and drink that tend to produce diarrhœa. Complete and sudden change of the mode of life is not advisable, and we should allow the moderate use of good red wine and strong beer

that is not too new and has not become sour. On the other hand, all excess should be carefully shunned. The foolish assertion, that these rules are useless, as many persons who are careful of their diet are taken sick, while others who live carelessly escape, should be answered with rational arguments, and persons who are susceptible to reason should be shown that no one knows that he is not already infected with cholera, and that the impending attack will certainly have a very severe course, if some other injurious influence besides the cholera-poison be acting on the intestinal canal. Lastly, the patients should be advised to send for a physician as soon as they are attacked by a diarrhœa, and to remain in bed till the physician comes, to drink a few cups of hot coffee or peppermint-tea, and to take some "cholera drops" which they should have on hand. It cannot be denied that energetic diaphoresis occasionally averts an attack of cholera. At least, in every cholera epidemic, we see persons that have been attacked by copious diarrhœa, great debility, cramps in the legs, and even vomiting, and who, on account of these symptoms have drunk large quantities of hot liquids (usually coffee with rum), buried in the bed-clothes and reeking with perspiration, while the passages, which were often discolored and beginning to resemble rice-water discharges, and the vomiting also, have ceased. Experience also teaches that in such cases, if the sweating be arrested too soon, a true cholera attack not unfrequently comes on, and that it is well not to let a cholera patient leave his bed till he has had a formed stool. The cholera drops, usually named after some well-known physician, that are sold by the apothecaries during cholera epidemics, consist of laudanum, generally with the addition of some ethereal tincture, which is superfluous and often detracts from the efficacy. Their use without medical advice should be recommended, because opium is one of the most efficient remedies against cholera diarrhœa, and because its success is the more certain the more recent the case. The so-called Russian cholera drops are particularly celebrated: ℞. tinct. valer. æth. ʒij; vin. ipecac. ʒi; tinct. opii ℈j; ol. menth. pip. gtt. v. ♏. S. twenty to twenty-five drops every hour or two.

While the most careful prophylactic treatment often fails, we are still less able to fulfil the indications from the cause or from the disease, after cholera has once broken out. In almost every epidemic, especially toward its close, when the malignancy of the disease has abated, and the number of recoveries is greater than that of the deaths, certain *specifics* are recommended both by physicians and quacks. But their reputation has never lasted through the first weeks of a subsequent epidemic. Radix sumbul, carbo trichloratus, and other remedies recommended as panaceas in cholera, have very justly been

thrown aside. Hence we must content ourselves with striving to fulfil the symptomatic indications, and we shall do this the more successfully, the more closely we attend to and combat those symptoms on which the others depend. The symptomatic treatment which, in the first epidemics, consisted in attempts to elevate the fallen temperature by vapor-baths, and by having the patient drink hot teas during the algid stage, but not allowing them a drop of cold water, and in attempting to draw blood from all cases of cholera asphyxia, was certainly incorrect. Depression of the temperature of the body is a late occurrence in the series of symptoms induced by the cholera infection; warm tea, which is more readily vomited than any other drink, is not nearly so well borne as small quantities of cold water; venesection cannot raise the depressed action of the heart, on which the venous congestion depends. The symptomatic treatment of cholera requires, first of all, attention to the intestinal disease, the arrest of the acute catarrh and extensive transudation of serum from the intestinal capillaries, the source of all the other symptoms and of the danger. The second symptomatic indication is, to replace the water lost from the blood. If we succeeded in making a cholera patient sweat while the transudation into the intestines continued, we should injure him by the increased abstraction of water. Lastly, the third indication, which we must bear in mind from the first, is to combat the threatening paralysis of the heart. We shall not discuss whether opium (the most frequent prescription in genuine intestinal catarrh, and the last refuge in all other diarrhœas), besides retarding the movement of the intestines, also lessens the secretion of the intestinal mucous membrane, and the increased transudation from the intestinal capillaries; at all events, it owes its frequent use in cholera to its antidiarrhœic action. Even after satisfying themselves that, in many cases, opium has had no effect on cholera diarrhœa, most physicians seek its aid in new cases, because they have sometimes found it of undoubted benefit even in this affection. I fully agree with this treatment, and, before trying other methods, I give opium for cholera diarrhœa, although not in the form of cholera drops but in the shape of Dover's powder, or as tincture in mucilage, without the addition of any ethereal substance. If the patient has taken a number of doses of opium (half a grain to a grain) in the course of a few hours, and the diarrhœa has improved, it is well to continue it in smaller doses till a formed stool shows that the excessive transudation into the intestines has ceased. If, on the other hand, in spite of the repeated doses of opium, the diarrhœa continues or grows worse, if the patient collapses visibly, if his skin grows cool, and the dejections lose their color, I regard the continuation of opium as contraindicated; while, in such cases, I have had the best results from

cold compresses frequently applied to the abdomen, and from the administration of calomel (a grain every hour). In regard to the speedy favorable effect of this treatment, especially of the application of cold compresses to the abdomen, on most patients, in regard to its favorable influence on the entire disease, and the principles which induced me to employ it, I refer to my brochure already mentioned, "Die Symptomatische Behandlung der Cholera," Magdeburg, 1848, and would only mention that, in 1854, when *Pfeufer* was commissioned to instruct the Bavarian physicians in the treatment of cholera, he recommended my method as being the most successful, according to his experience. Nitrate of silver, which has been recommended by many persons, especially by *Levy* of Breslau, and which I have frequently employed, because, *a priori*, it seemed the most efficient, did not succeed with me. The second indication, to replace the loss of water from the blood by supplying water, is best attained by giving the patient small portions of ice-water, or small pieces of ice to swallow at short intervals. Large quantities of liquid, especially of warm drink, are usually vomited at once. At all events, since cholera patients have been almost universally allowed to drink cold water, they suffer less than when, in spite of the torturing thirst, they were allowed no drink, or, at most, warm tea. As the paralysis of the heart disappears, as the transudation from the capillaries ceases, and the normal functions of the stomach and intestines are restored, the circulation usually becomes normal at once, and, without the use of any stimulant, the action of the heart, even where reduced to a minimum, may become greater than normal within a few hours. But this does not prove that stimulants are useless or unnecessary in the treatment of cholera; in the earlier epidemics, they were almost exclusively employed. As soon as the pulse grows small and the patients are evidently in collapse, stimulants should be given from time to time, to try and prevent complete paralysis of the heart, until the termination of the acute disease in the intestines. Among the stimulants, champagne that has stood on ice is preferable to most others, especially to the ethereal oils, carbonate of ammonia, etc., because, along with its stimulant action on the nervous system, it has no irritant effect on the gastric and intestinal mucous membrane. In poor practice, rum or arrack, diluted with water, is best. In some cases it is well to alternate the administration of ice and ice-water with a few cups of hot strong coffee. It is often vomited again, but frequently not till the pulse has become fuller, and the temperature somewhat elevated. If the evacuations upward and downward have ceased, while the continuance of the symptoms of collapse shows that paralysis of the intestinal muscles, and not arrest of the transudation, has induced this change in the disease, stimulation

is the proper treatment, and the return of the evacuations is the best evidence of its success. Frictions of the skin with tincture of mustard often relieve the painful cramps in the muscles; but I would warn against the very common application of mustard-plasters. For, even if they be left on a long while, the patients rarely complain of their burning, and the attendants are so excited by the fearful appearance of the patient that they lose their presence of mind, and forget the sinapisms, and I have seen them left on half a day, and during convalescence the patient has been troubled by obstinate and painful inflammations of the skin induced by the mustard. Of course, nourishment cannot be given to cholera patients during the actual attack; but, even after the attack is over and reaction has begun, we should be very careful about the food, and in order to protect the diseased intestine from injury, we should give nothing more irritating than diluted milk, meat-broth, and biscuit. Nutritious and solid food should not be allowed till pulpy and consistent stools appear. Infringement of this rule is generally severely punished.

No general rules can be given for the treatment of the stage of reaction, and still less for the sequelæ of cholera comprised under the name of cholera typhoid, for the treatment must be based on a careful analysis of the symptoms in each case. The former custom of bleeding for violent reaction should be discontinued. If there be evidences of great fluxion to the brain, we should apply ice compresses to the head and leeches behind the ears. But we must take care not to mistake the hydrocephaloid that often occurs in children after an attack of cholera with hyperæmia and œdema of the brain. If the severe symptoms of cholera typhoid depend on uræmic intoxication, we may employ the treatment already laid down, little as is the prospect of success. If the attack be followed by an asthenic fever with typhoid symptoms, and the belly be puffed up and painful, and thin but colored and badly-smelling evacuations are passed from time to time, we may cover the abdomen with cataplasms, and order small doses of calomel and opium. In the same way, when treating inflammations of the different organs occurring after cholera, we must bear in mind the exhaustion of the patient.

CHAPTER XVI.

BLOODY FLUX—DYSENTERY.

Etiology.—Dysentery is an infectious disease (when treating of diseases of the intestinal canal, in the first volume, we spoke of "catarrhal dysentery," which does not depend on infection); but it differs from typhus, and other infectious diseases, in that the infection with dysenteric poison induces perceptible pathological changes in the in-

testinal canal only. The anomalies observed in other organs, and in the blood, during dysentery, as well as the more or less severe fever accompanying the disease, are secondary symptoms, induced by the intestinal affection. Dysentery is thus closely allied to cholera, where the infection also causes, first, a severe disease of the intestinal mucous membrane, and, secondarily, as a result of this primary disease, changes in the composition of the blood, in the circulation, and in the nutrition of the various organs. Of course, the intestinal affection is not the same in dysentery as in cholera, and consequently its influence on the blood differs from that of cholera.

Dysentery poison cannot be directly observed, as an organic, living substance, any more than the poisons inducing other infectious diseases can, but the reasons so often repeated, especially when speaking of typhus, induce us to refer dysentery also to an infection of the body by a certain species of low vegetable organism, and to speak of a "dysentery germ," as we have already spoken of a "typhus germ" and a "cholera germ." From this point of view, we may, to some extent, understand the facts which have been determined by thorough observation concerning the spread of the disease.

Dysentery results, although not exclusively, from a miasm; or, in other words, the dysentery germ grows, flourishes, and increases, outside of the human body, and persons staying near its locality are in danger of being attacked by it. The circumstances favorable to the increase and propagation of dysentery poison, among which a high temperature and a certain amount of moisture are prominent, exist in the tropical regions; there the disease is endemic through large portions of country. According to the classical work of *Hirsch*, in Europe only the peninsulas, as the south of the continent, and the islands about them, constantly offer such favorable conditions for the increase of the dysentery germ as to cause the disease to be endemic there. But, through almost all Europe, the conditions for the increase and propagation of dysentery, which is endemic with us also, are occasionally so favorable, especially late in the summer, that the disease becomes epidemic. The circumstance that dysentery is not endemic or epidemic in all regions where high temperature and moisture constantly exist, justifies the conclusion that these are not the only things necessary for the growth of the germ, or else that it is not so widely spread as to be found everywhere that conditions favorable to its development exist. The coincident epidemic or endemic occurrence of dysentery and intermittent is frequent, but not at all constant, according to the recent observations of *Hirsch*. Dysentery exists where the requirements for malaria, marshes, etc., are not present. It attacks the open country oftener than the city.

The dysentery germ appears to reproduce itself always, or under favorable circumstances, in the body of the infected person, and it would seem that the dejections of the patient contain the contagion thus formed, or its components; for, while it has not been proved that one person catches dysentery from another, it is more than probable that the disease may be communicated to healthy persons through the dejections of dysentery patients, or by the night-stools, bed-pans, or enema syringes that have been used by them. This causes dysentery to resemble cholera, while it speaks against the asserted connection between it and malaria. Why should not the same or similar influences, such as high temperature and moisture, favor the development of various specific low organisms, just as it does the increase of different varieties of higher plants and animals?

Catching cold, getting wet, great fatigue, the use of unripe vegetables, and other injurious influences, have been advanced as causes of dysentery, and it cannot be denied that persons exposed to these influences are more readily affected than others. Nevertheless, infection with a specific poison is the sole cause of this disease, and the part that the above influences play in the etiology is only to render the organism more sensitive to the action of the poison; in other words, they increase the predisposition to dysentery.

Anatomical Appearances.—The anatomical changes found in the intestines of dysentery patients, on autopsy, are a type of diphtheritic inflammation. The diseased portions of mucous membrane are infiltrated with a fibrinous exudation, and, as a result of their compression by the exudation, are necrosed and changed to a slough. If the slough be cast off, a loss of substance in the mucous membrane remains. According as this loss of substance is superficial and of slight extent, or deep and extensive, the destruction of the mucous membrane is, or is not, capable of complete repair. In the former case only can the intestine become perfectly well, while in the latter, in place of the membrane destroyed, there is a callous cicatricial tissue, which not unfrequently constricts the intestine.

In the mildest grades of dysentery we find the mucous membrane of the large intestine, especially the summits of the folds projecting inward, deeply reddened by ecchymosis and injection, and to some extent infiltrated by a grayish-white, soft exudation, covering the epithelial coating. In these cases, it looks as if the diseased part were covered by a bran-like coating; but if we scrape off this coating with the handle of a scalpel, there remains a superficial loss of substance, which shows that the exudation did not lie on the mucous membrane, but entered into it. The submucous connective tissue is infiltrated with serum, and swollen. The serous coat appears cloudy and dull

from œdema. This change occurs chiefly, and generally exclusively, in the large intestine, and it is rare for the dysenteric affection to extend to the lower portion of the small intestine. In higher grades of the disease, more glutinous, or more hard, membranous gray-white layers cover large portions of the interior surface of the intestine; they are removed with difficulty, and only along with the mucous membrane. If they are already detached, the submucous tissue lies exposed. The whole intestinal wall appears thickened by the excessive œdema of the submucous tissue, and of the muscular and serous coats, but certain parts, corresponding to the spots formed by the exudation, are especially swollen, so that they form nodular prominences on the inner surface of the intestine. When dysentery is of this high grade, the serous coat of the intestine usually participates in the inflammation, and is covered with a thin layer of fibrin, which unites it to the surrounding parts. The diseased intestine is evidently dilated, and is filled with epithelial masses, shreds of exudation, and with an albuminous fluid mixed with more or less blood; it usually contains no fæces. In the highest grades of dysentery, according to *Rokitansky*, "large portions of the mucous membrane are changed to a black, rotten, friable, charred mass, which subsequently is not unfrequently thrown off and passed as tubular pieces; the submucous tissue either appears infiltrated by charred-looking blood and by a bloody serous fluid, or else pale, and the blood in its vessels is consumed to a black, stiff, powdery mass; but later, as a result of the throwing off of the dead parts by a reactive inflammation in the deeper layers, it appears infiltrated with pus. Besides a dirty-grayish discoloration and loss of lustre, the peritoneal coat is in some places injected by dilated capillaries, and covered by a brownish, discolored, ichorous exudation. The foul-smelling intestine, containing a blackish-brown fluid, like coffee-grounds, is either in a state of passive dilatation, or it is collapsed, and, when the process has lasted a long time, the muscular coat is shrunken, pale, faded, and readily torn."

The glands of the mesocolon belonging to the diseased portion of intestine are more or less vascular, swollen, and relaxed. The liver is hyperæmic, and in the malignant dysentery of the tropics it is not unfrequently the seat of abscesses, which are doubtless due to the intestinal veins carrying ichor or emboli from the diseased intestine to the liver.

If death do not occur at the height of the disease, the dysenteric process either subsides, or a slow inflammation ("chronic dysentery") remains, which subsequently carries off not a few of the patients. In the former case, the edges of the loss of substance are approximated, by the contraction of the connective tissue which forms their base, till

they finally come in contact, if the loss has not been too great. Then, as in cicatrizing ulcers of the stomach, there is sometimes stricture, sometimes not. If, on the other hand, the opening be very large, its edges do not come together, and we find more or less extensive patches of the inner surface covered, not by mucous membrane, but by callous connective tissue. According to *Rokitansky's* apt description, this tissue not unfrequently forms "fibrous bands and striæ, which project in the shape of seams into the intestine, cross each other in various directions, and often form valvular or ring-shaped duplicatures in the intestine, thus inducing a very peculiar stricture of the colon." If, on the other hand, the intestinal disease takes a chronic course or tends to recovery, the losses of substance become chronic ulcers, and those changes occur in the intestine which we described as follicular ulceration. In the thickened, strongly pigmented, mucous membrane, the inflamed follicles become ulcers, at first round, afterward irregular; occasionally, also, in the thickened submucous tissue, abscesses and fistulous passages form, and subsequently perforate the muscular coat and induce peritonitis or abscesses about the rectum.

Symptoms and Course.—Occasionally, premonitory symptoms precede the actual outbreak of the disease for several days; these consist in an undefined constitutional disturbance and irregularity of digestion, especially loss of appetite, thirst, slight colicky pains, and inclination to diarrhœa. The commencement of the disease is rarely characterized by a chill, and not often by rigors even, or other symptoms of fever. But in most cases dysentery begins with an apparently innocent diarrhœa, during which the fæces passed are not suspicious-looking, which is preceded by very moderate colicky pain, and is accompanied by very little, if any, of the tenesmus which afterward becomes so painful. But the more frequently the passages succeed each other, the more severe and continued become the colicky pains (tormina ventris) which begin some time before each evacuation, and shortly before its occurrence attain great severity. The evacuations are accompanied by a very torturing and painful bearing down of the rectum, to which is often added strangury. In spite of the severe and long-continued straining, proportionately slight, non-feculent, mucous, gray-colored masses (dysenteria alba) or muco-bloody masses (dysenteria rubra), and occasionally pure blood, are evacuated. In some cases a few hard scybala are from time to time passed with the mucous or muco-bloody masses. Immediately after an evacuation the patient feels relieved, and usually has pain only on hard pressure against the abdomen, especially in the region of the colon; but soon, often even in a few minutes, the tormina begin again; the patient writhes and groans, and, when the pains have attained the highest grade, tenesmus

recommences, and again a small quantity of dysenteric dejections of a sickening odor is passed. Sometimes this scene is repeated twenty or thirty times in twenty-four hours. If at first absent, febrile symptoms always come on in the course of the disease. Where the intestinal lesion is moderately intense, the fever has an erethetic or synochal character; the pulse is moderately frequent, full, and hard. But in the highest grades of the disease the fever acquires an asthenic character very early; the pulse becomes small, and very frequent. According to the character of the fever, dysentery has been divided into inflammatory, adynamic, and putrid, or typhous. This classification corresponds very nearly with the different grades that we described above. Even in the mildest grades, and when the fever is moderate, the patients are greatly run down by the loss of albumen, the pain, and the loss of sleep; they become pale; the pulse, at first full, grows small; there is great mental depression; the dulness and want of spirits are very marked. If we filter the dejections, and add nitric acid to the filtrate, we shall find that the albumen in it is sufficient to stiffen almost the whole contents of the reagent-glass, even where the dejections scarcely have a reddish tinge, and only solitary blood-corpuscles are found under the microscope. This great loss of albumen also explains why, in favorable cases—where, after four to eight days, the tormina and tenesmus grow less and gradually disappear, and where, often by the end of the first, or the beginning of the second week, the masses passed again become feculent—the convalescence is almost always slow. The character of the blood of a convalescent from dysentery is, as *Schmidt* aptly remarks, very similar to that of a person with *Bright's* disease; and I can confirm, from my own experience, the observation that general dropsy follows slight cases of dysentery far more frequently than it does other diseases of equally short duration.

In the *higher* grades of dysentery, the evacuations succeed each other at very short intervals, the colicky pains scarcely cease, and occasionally become unbearable; the abdomen is sensitive to even a light pressure. The tenesmus also is more continued and severe than in the milder forms. The dejections contain a great deal of blood, numerous flocculi and shreds, and occasionally large membranous masses. In many cases large quantities of pure blood are evacuated. At first the pulse is more frequent and full; later it becomes very frequent, while its fulness usually diminishes rapidly. There are also great constitutional disturbance, loss of appetite, dry tongue, deep physical and mental depression, and frequently dulness of the mind and slight delirium. If the disease runs a favorable course, the symptoms gradually subside, the intervals between the evacuations become longer, the dejections again grow brownish and feculent; the epithelial and exu-

dation masses, as well as the blood, which long continues to be mixed with them, diminish; the pulse rises, the tongue grows moist, the mind clearer; but convalescence is always slow, and, in the most favorable cases, weeks elapse before the patient can leave his bed. If the disease is to prove fatal, the pulse becomes smaller, the apathy increases, consciousness is lost, the complaints of pain and tenesmus cease, the evacuations are involuntary, and the patient dies, as in other rapidly-exhausting diseases, of general paralysis. If the disease pass from the acute to the chronic form, as is very often the case in the higher grades, the fever disappears, and we have the symptoms of follicular ulceration of the intestines. Diarrhœa generally alternates with constipation; occasionally normal fæces, with muco-bloody masses clinging to them, at other times only a puriform fluid from the ulcerating mucous membrane, is evacuated; the patients become very much emaciated, and, after languishing for months, usually die of marasmus and dropsy. If the losses of intestinal mucous membrane heal, leaving a cicatricial stricture, the symptoms of protracted dysentery of high grade are followed by those of stricture of the intestine. For the rest of his life the patient suffers from habitual constipation, and the various inconveniences accompanying this state. On carefully inquiring into the causes of chronic abdominal diseases, we can frequently trace them to a dysentery that existed several years previously.

In the highest forms of dysentery, the putrid or septic forms of authors, after the disease begins as above described, the passages assume a discolored, brownish-red or blackish color, and a carrion-like odor; and large, black, sloughy shreds of mucous membrane are not unfrequently mixed with them. The pulse soon becomes small and very frequent, the extremities cool, and the body burning; the patients are collapsed, the countenance distorted, the tongue and gums are covered with dry sordes, and the mind is very dull. Tormina and tenesmus cease very early in the disease; the discolored, thin, fetid passages are passed involuntarily, owing to the relaxation of the sphincter, and they excoriate the parts with which they come in contact. In such cases, to the symptoms of adynamia are often added those of an acute hæmorrhagic diathesis, bleeding from the nose, petechia, etc. During the first days even, the patient may die of septic dysentery; and recovery is very rare in this form of the malady, which occurs chiefly in camps, besieged towns, or under other unfavorable conditions. Dysentery patients with the higher and highest forms of the affection do not often die of peritonitis or pyæmia; in the tropics not a few probably die later from the hepatic abscesses which develop during the disease.

Treatment.—Prophylaxis demands that the circumstances which experience shows to favor the development and propagation of dysen-

tery poison be removed as much as possible; the rules for obtaining this end are, according to what was said above, partly the same as those treated of in the prophylaxis of intermittent fever, partly those of typhus and cholera. Since the dejections of dysentery patients are very probably the bearers of the poison reproduced in the infected organism, prophylaxis further requires that the bed-pans, enema syringes, etc., of dysentery patients should never be used for other persons, and that the dejections should not be thrown into the common privy, but into a separate pit, and disinfected with a solution of sulphate of iron. Lastly, prophylaxis requires the avoidance of all those causes which increase the tendency to dysentery by rendering the body more susceptible to the action of the poison. Little probability as there is of a person acquiring dysentery by eating unripe fruit, sleeping on the wet ground, etc., if he be not at the same time exposed to the dysentery poison, it is still certain that, during an epidemic, these causes favor the outbreak of the disease.

We cannot fulfil the causal indications, or those from the disease in dysentery, as we know no antidote that counteracts the poison. The attempt to arrest the disease by active treatment with bleeding, emetics, purgatives, large doses of opium, etc., has very properly been abandoned, and, at present, the treatment is limited to combating the symptoms. But the symptomatic treatment of dysentery only promises good results, if we bear in mind the dependence of the diarrhœa, tormina, tenesmus, and other symptoms, on a diphtheritic inflammation of the intestinal mucous membrane. If we do not attend to this fact, but give opium to arrest the diarrhœa even in cases where hard masses of fæces are collected above the inflamed portion of intestine, we shall render the disease worse, for impacted and decomposed fæces alone are enough to excite a diphtheritic inflammation. In the *milder* grades of dysentery it is well to begin the treatment with a mild laxative, such as castor-oil or decoction of tamarinds, and to return to this remedy every time that the dejections contain no fæcal matter for a day or two. The reason for this treatment has just been explained. It is only advisable to give an emetic, of ipecac. or tartrate of antimony, in cases where the stomach is filled with undigested substances. Even in the mildest grades of dysentery, the patient should carefully keep in bed, and eat nothing solid, but live on soup diet. If he be strong and full-blooded, mucilaginous water-soup is sufficient; but, if he be weak and anæmic, it is well to attend to keeping up the strength from the first and to advise concentrated meat-broths. Most patients are relieved by the application of warm poultices to the abdomen. If the tormina be very severe, and be not relieved by the cataplasms, or if the abdomen be unusually sensitive to pressure, we shall find great

benefit from the application of leeches to the abdomen (in adults ten to twenty), and allowing the bites to bleed for a time under the poultices. Internally we may give an emulsion, and at evening a moderate dose of opium. This treatment is enough for many cases of mild dysentery; but in others, in spite of it, the passages increase in number, the tormina and tenesmus are more severe, and the fever augments greatly. In such cases, as well as in the *higher* grades of dysentery where local abstraction of blood is almost always indicated, the administration of calomel with opium is the most trustworthy treatment. It is customary to give one grain of calomel with a quarter of a grain of opium every two hours; and I think this treatment, especially when combined with five to ten grains of Dover's powder at bed-time, deserves the preference over large doses (gr. x) of calomel, which have also been recommended. If salivation result from the continued use of calomel, we must stop it, and continue the opium alone, then it is best to give the tincture in mucilage, or in a weak infusion of ipecac. In just such cases a combination of opium and acetate of lead is highly spoken of. But I always consider it best, besides the small doses of opium given during the day, to give a full dose at night. If this treatment also fail, we have still less to expect from the internal administration of nitrate of silver and the vegetable astringents, especially tannin. According to my experience, the employment of the latter remedy in the form of enema, which is praised by many authors, is objectionable on account of the difficulty of giving a clyster when the tenesmus is so great, and on account of the increase of the tenesmus caused by the most careful injection as well as by the medicament itself. Moreover, if the remedy is to come in contact with the whole of the diseased surface, the enema must be very large; the contents of a simple enema syringe do not pass much above the rectum. In the *highest* grades of dysentery, treatment is almost always without benefit. The great prostration of the patient, the threatening paralysis forbid the abstraction of blood as well as the administration of calomel and opium, and we must limit ourselves to maintaining the strength of the patient as well as possible, and preventing general paralysis, by the administration of tonics and stimulants. In chronic dysentery, especially when the tenesmus has abated, enemata of solutions of nitrate of silver or sulphate of zinc deserve most confidence. If there be no collection of fæces above the seat of disease, or if the muco-purulent and bloody masses are accompanied by the passage of thin fæcal matter, we may give astringents internally also, and in such cases I prefer catechu (℥ ij to ℥ vj water and ℥ ss gum-arabic, a tablespoonful every two hours) to tannin and nitrate of silver, as it is doubtful if the latter reach the intestines in a very efficient form. If

stricture of the intestine remain after dysentery, it is to be treated as previously described.

CHAPTER XVII.

TRICHINA DISEASE—TRICHINOSIS.

THERE may be some doubt in what division of special pathology trichina disease should be considered; whether it should be treated of among the diseases of the stomach, as the first seat of the disease is there, or among the diseases of the organs of motion, as the trichinæ pass from the intestinal canal into the muscles; or among the infectious diseases. In the present edition I have decided to treat of trichinosis among the infectious diseases, as I here uphold the dependence of the infectious diseases on the presence of a lower organism, more strongly than I did in former editions. The origin of trichinosis from a contagium vivum is proved. The symptoms have the greatest resemblance to those of the infectious diseases. Even the period of incubation is common to both. We may go still further and say that there is far less doubt about trichinosis being an infectious disease than about any other disease belonging to this class.

ETIOLOGY.—For more than twenty years numerous punctate, white bodies had occasionally been seen in the muscles, on autopsy, which the microscope showed were small cysts containing a thread-like worm, wound up spirally. This worm, the trichina spiralis, showed no sexual organs, and it was altogether obscure whence it came, how it reached the muscles, and whether it was capable of further development. We have already shown that the supposition, according to which the muscular trichina was the undeveloped state of the tricocephalus dispar, was soon found to be erroneous. From experiments, by *Virchow*, *Leuckart*, and others, of feeding animals with flesh containing trichinæ, it was shown that the muscular trichinæ become free in the stomach and intestines of the animals fed, during the digestion of the meat; in a few days they attain the length of three or four millimeters, and form male and female perfect animals, intestinal trichinæ. In the female, which is far more numerous and about two-thirds larger than the male, innumerable eggs develop, and from these young ones escape, living, from the body of the mother, even in from five to eight days, and move freely in the intestines. The young brood of the intestinal trichinæ now soon perforate the wall of the intestine, part of them passing into the abdominal cavity, part between the folds of the mesentery to the spinal column, thence to the diaphragm, abdominal muscles, and following the intermuscular connective tissue to the other muscles of the body. The number of these wandering trichinæ is innumerable. They

are particularly numerous about the ends of the muscle, where it becomes tendinous, probably because their further progress is obstructed at these points. Trichinæ have a particular preference for the muscles of the loins, diaphragm, the intercostal and cervical muscles, and for those of the eye and larynx. In the extremities, as a rule, "the further from the trunk, the fewer trichinæ in the muscles" (*Reny*). In the human being, at least, they seem never to enter the muscles of the heart. The wandering of trichinæ induces parenchymatous and interstitial myositis. The free, emigrating trichinæ, which gradually attain the length of one-third of a line, are not visible to the naked eye. At the point where they finally become attached, the irritation they induce leads to a development of the sarcolemma, and thus to the formation of a capsule, which is egg or lemon-shaped. This capsule grows thicker; calcareous salts are deposited in it, and it is the trichinæ enclosed by such calcified capsules that are visible to the naked eye. At least two months are required for the formation of a perfect capsule.

The etiology of the trichina disease in animals is still obscure. Of course the solution of the question, whence come the trichinæ in pork? is very interesting, and of great practical importance. It is probable that they acquire them chiefly or solely by eating trichinous rats. It is well known that pigs not unfrequently eat live and dead rats, and it is also known that rats not only frequently have trichinæ, but that they often die of them.

In the human being the trichina disease results solely from eating trichinous pork. The raw flesh is the most dangerous, hence the disease is proportionately more frequent and more severe in those who have the bad habit of eating raw meat than in others. More frequently than in the raw form, pork is eaten prepared in a manner that does not kill the trichinæ. Even large pieces of well-boiled or baked pork, which do not show any red spots on being cut, and into which we may easily pass a fork without a feeling of grating, never contain trichinæ. But they may exist in roast pork, whose external portion only has been exposed to a temperature of 55° R. [156° F.], while the inner part has not been heated so much, looks bloody, and is somewhat hard and coherent. And even in small pieces of meat, and in the various forms of sausages, if they be cooked for only a short time, the trichinæ remain alive in the parts distant from the surface. Trichinæ are found especially often after the use of so-called fresh blood and meat sausages, of meat-balls, and similar preparations. Long-continued salting (pickling) of pork, without the addition of water, kills the trichinæ even in large pieces; on the other hand, numerous trichinæ may survive in pickled meat that has only

remained a short time in a weak solution of salt. Smoking the meat only kills those trichinæ that are near the surface. The so-called quick smoking, where the ham is painted with pyroligneous, acetic acid, or creasote, and left in the smoke only a short time, or not at all, affords the least protection. Poisoning by raw hams, chitterlings, brain pudding, chopped sausage, and other slightly-smoked varieties of sausage, is often seen. The fact that smoked trichinous flesh is repeatedly eaten without injury, is partly because it has been carefully pickled for a long time before smoking, and partly because the meat in question had been kept for a long time, and completely dried, which also kills the trichinæ. Very few cases of encapsulated trichinæ, and not a single one of acute trichina poisoning, have been observed in southern Germany; this is because the people there dislike raw flesh, even when pickled and smoked.

Since all persons who eat living trichinæ are not equally affected, and as the severity of the disease is not by any means always in proportion to the number of trichinæ probably introduced into the stomach, we may speak of a greater or less predisposition to trichinosis. But the causes on which this predisposition depends are not yet known. We can only assert that it is connected with the condition of the gastric and intestinal mucous membrane, or with the character of the contents of the stomach and intestines, and that, after the use of trichinous meat, severe diarrhœa, by which the still undigested portions of flesh, with the trichinæ contained in them, are evacuated, is to be regarded as favorable. Children have a certain immunity to trichinosis; at least children recover from trichina poisoning, from which they certainly are not exempt, more readily than adults, perhaps because they do not digest a part of the meat.

Anatomical Appearance.—On autopsy of the lower animals that have been poisoned with trichinæ, if they have died or been killed during the first weeks, besides the numerous intestinal trichinæ and young ones that have already entered the muscles, we find in many cases the remains of enteritis and peritonitis. On the other hand, on autopsy of human beings who have died in the first week or two after accidental poisoning by trichinæ, we never find exudation in the intestine or peritonæum, but only the signs of a more or less intense intestinal catarrh, and often a decided swelling of the mesenteric glands. This is doubtless because, on intentionally poisoning, a much larger number of trichinæ is given them than is swallowed by a person accidentally so poisoned, consequently a much larger number of the trichinæ pass through the intestinal walls, and the lesion caused by them is far more intense. After the fifth week, distinct signs of interstitial and parenchymatous inflammation appear in the muscles as fine, grayish-

red striæ. The microscopic examination of such spots shows that in them the muscular fibrillæ have broken down to a granular detritus mass, while the interstitial connective tissue is increased by fresh proliferation. The number of trichinæ in the muscles is the greater, and the parts affected more extensive, the longer the disease has lasted. In protracted cases we find even the muscles of the extremities containing quantities of trichinæ. This circumstance, and the experience that even in the seventh and eighth week we find living intestinal trichinæ, filled with eggs and embryos, render it not improbable that the intestinal trichinæ bear young not only once but repeatedly, and that they pass into the muscles at different times. The other changes found in the bodies of trichina patients are not pathognomonic, and correspond with those found in the bodies of persons who have died of other diseases, accompanied by high fever and rapid exhaustion. According to *Cohnheim*, even the muscles have no constant and characteristic appearances, except the above-mentioned anomalies, and a certain density and toughness, but in regard to their color and moisture vary greatly, just as they do in typhus. In many cases there are signs of extensive bronchitis, and of hypostasis in the lungs, or pneumonic infiltration, and sometimes thromboses in the veins. Lastly, when the disease has lasted a long while, and the fever has been very severe, in some bodies we find the parenchymatous degenerations of the liver, kidneys, and heart, which we have already mentioned.

Symptoms and Course.—Since *Zenker*, in 1860, made the important discovery that the entrance of trichinæ into the human body caused a severe and even fatal disease, a number of violent epidemics of trichinosis have been so carefully observed and described, that the symptomatology of the newly-discovered disease is now as well determined as that of most other maladies that have been known a long time.

Before the trichinæ which have entered the stomach are set free by the digestion of the flesh in which they are embedded, the patients have no trouble. In some cases, which *Renz* calls insidious trichinosis, even when the trichinæ have become free, when they have mated and are rearing their young, and when the young brood perforate the intestinal canal, there are no signs of an intestinal disease. Such patients have a good appetite, regular bowels; they feel fatigued and depressed, and complain of travelling pains, and a certain stiffness in the limbs, it is true, but they can go out and attend to their business. Gradually the vague pains are localized in certain muscles: these swell, become hard and rigid; œdema, fever, and other symptoms characteristic of trichinous myositis are developed. It is most probable that, when the disease runs this course, only a few trichinæ have reached the stom-

ach, or remained there, and that consequently the development of the intestinal trichinæ and their passage through the walls have only slightly interfered with the functions of the stomach and intestines, while successive broods and repeated emigrations of young trichinæ have filled the muscles with the parasites. It is difficult to explain the occasional sudden change of insidious trichinosis into dangerous forms, unless it be due to an extensive emigration of a new generation of trichinæ into the respiratory muscles.

This insidious commencement of the disease, with its total absence of gastric disturbance, is in striking contrast with the very troublesome intestinal symptoms observed at the commencement of some cases. The first cases in the famous Hederleben epidemic were regarded as cholera, because the patients were attacked with severe vomiting and purging, which could not be checked. Three of them died on the sixth day of the disease, with the symptoms of paralysis and thickening of the blood. For the diagnosis between "trichinous cholera" and Asiatic cholera, and cholera morbus, *Kratz* and *Ruprecht* lay particular stress on the peculiar stretching, muscular pain, which is located chiefly in the flexors of the extremities, and is increased both by movement and pressure. The commencement of trichinosis with cholera symptoms, which is not frequent, shows that an unusually large number of living trichinæ have reached the stomach, and that consequently the gastric and intestinal walls have suffered more than usually. This idea is supported by the fact that cholera symptoms have hitherto been observed only in cases of poisoning with raw meat.

The absence of all intestinal symptoms, as well as severe attacks of vomiting and purging, is only exceptionally observed after the use of trichinous flesh. Far the greater number of patients complain in a few hours, or not till a few days after the poisoning, when the young brood has been hatched, of severe pressure in the stomach, of eructation and nausea, combined with a feeling of great heaviness and depression. There is almost always diarrhœa, the passages being at first brownish, subsequently yellow, thin, and accompanied by more or less severe colicky pain. These gastric symptoms are soon accompanied by those of the entrance of the trichinæ into the muscles, vague pains, and a feeling of stiffness, as well as a peculiar œdema of the face affecting chiefly the eyelids, in which the conjunctiva also participates occasionally, so that there is chemosis. The movements of the patient are now sometimes very much impaired, partly because their muscles become more rigid and less supple, partly because every attempt to stretch them is very painful. The different muscles swell considerably, become tense and as hard as caoutchouc, just as in the rigor mortis. According to *Cohnheim's* description, in severe cases the constant and

characteristic position of the patient is as follows: "He lies on the back with the shoulder and elbow-joints sharply bent, and the hands slightly flexed; on the other hand, the knee and hip-joints are but slightly bent, or nearly straight; so that the patient is unable to lift the arm, extend the forearm, sit up, or bend the knees." *Colnheim* explains this position correctly, by ascribing it to the patient's attempt to assume a position where the different groups of muscles are the least extended; with the swelling of the muscles there is œdema extending from the arms toward the hands, from the thighs toward the feet, but not affecting the scrotum or labia. Besides the above symptoms, from the third to the fifth week of the disease, there are frequently attacks of severe dyspnœa, which subside again in the sixth week; they doubtless depend on trichinous disease of the respiratory muscles. Where the disease attacks the muscles of the glottis, the voice is sometimes lost; sometimes affection of the masticatory muscles causes trismus; participation of the glossal and pharyngeal muscles induces impaired movement of the tongue and dysphagia. Trichinosis is accompanied by high fever, with slight morning remissions. Although this fever is unmistakably not of zymotic origin, but depends on the extensive disease of the muscles, and is to be regarded as a symptomatic inflammatory fever, it very closely resembles the fever observed in the course of typhus and other infectious diseases, and occasionally the temperature curves can scarcely be distinguished from those of typhus. The evening temperature sometimes reaches 106°, that of the morning remains somewhat lower. The bodily temperature does not become normal for a long time, often not till the sixth or seventh week. The frequency of the pulse corresponds with the temperature, and in severe cases reaches 120 to 140 beats a minute. Copious perspiration, with a miliary eruption, is somewhat characteristic of the fever accompanying trichinosis. The constitutional influence of trichinous fever, the effect that it has on the sensorium and other functions, is just the same as that from great elevation of the bodily temperature in other diseases. The pulse becomes small and weak, the thirst painful, the tongue dry, the patients sink into apathy, or become delirious; occasionally there are twitching and trembling of small groups of muscles, bed-sores over the sacrum, and with these symptoms the patients may die of exhaustion. The usual symptoms of trichinosis are more or less modified by the occurrence of extensive bronchitis, hypostasis, or pneumonic infiltration; but the pneumonic infiltrations alone (which are frequent) betray their presence by subjective as well as objective symptoms, by piercing pain in the side, cough, dyspnœa, etc. If the disease takes a favorable course, as not unfrequently happens in severe cases, the muscles gradually become less rigid and painful, the bodily

temperature and the pulse lower, the perspiration less copious, the thirst abates, appetite returns, but the exhausted patients are confined to bed for a long time, and regain strength very slowly. Just as after other severe diseases, œdema often occurs during convalescence, affecting even the scrotum and labia.

Treatment.—*Prophylaxis.*—When the flesh of a slaughtered hog is carefully examined microscopically, and no trichinæ can be found, it may be used, even raw, without danger. There is no doubt that, if competent microscopists examined the flesh of all the pigs killed, new epidemics of trichinosis might be avoided with certainty. I do not think it would be at all difficult, either in the city or country, to find competent persons, who could be so instructed, by microscopical courses and popular theoretical teaching, that they could tell certainly whether specimens of meat referred to them contained trichinæ or not. My brother, by his trichina catechism, and by practical courses, has fully qualified a large number of the laity for the microscopical examination of pork for trichinæ. Nevertheless, in opposition to celebrated authorities, I must say that I regard the introduction of an obligatory microscopical examination of meat as no guaranty against new epidemics of trichinosis. I have not sufficient trust in the conscientiousness of the persons to whom this would be intrusted, especially in the rural districts, to believe that, after they had examined for trichinæ in vain for years, they would continue their examinations with the necessary exactness. It is evident that a microscopical examination, if not carefully conducted, might prove dangerous, because persons relying on it might carelessly eat uncooked meat. The only certain protection against trichina-poisoning is, to eat no pork that has not been prepared in a way that would kill any trichinæ present. Whoever wishes to eat uncooked pork, smoked ham, sausage, etc., should previously inform himself that the pig from which they came contained no trichinæ. I advise my students to warn their patients (even those living in places where the flesh is examined) against the use of all dishes prepared from pork, which we mentioned under Etiology as being dangerous.

Treatment of the Disease.—From what we have said of the tenacity of life of muscular trichinæ, there is little hope of finding a remedy by which they may be killed without injury to the patient in whom they exist. *Friedreich's* proposal, to use the very bitter nitropicrate of potash in trichinosis, has not proved serviceable; it certainly permeates all the tissues of the body, but the views regarding its anthelmintic action differ. It is also doubtful whether benzine, as recommended by *Mosler*, has any effect. There is far more hope that, some time, we shall find a remedy to kill intestinal trichinæ, or remove them from the bowels. This indication exists, not only in recent cases, but must be remem-

bered later in the disease, since it is proved that, after weeks, living intestinal trichinæ, filled with embryos, may exist in the bowels, and consequently it is not improbable that there may be new emigrations of trichinæ into the muscles. The presence of diarrhœa should not deter us from beginning the treatment with a few doses of calomel or castor-oil, and, if necessary, repeating this treatment occasionally, as advised by *Ruprecht*. It does not seem to me to be proved that benzine, of which *Mosler* gives ℥ j to ℥ ij in gelatin capsules, is inefficacious against intestinal trichinæ, so that we should cease using it. The rest of the treatment is symptomatic. For the fever, quinine is advised ; for progressive prostration, stimulants ; for the subsequent anæmia, the preparations of iron. *Mosler* proposes, as the most efficacious remedy for the painful swelling of the muscles, the use of long-continued warm baths.

[The origin of the trichinæ in swine is an interesting question. The assertion that the disease originates in the rat is doubted, for it is said that trichinous rats come from slaughter-houses, where they may have fed on hogs' flesh. It is at least as probable that trichinæ are original with swine as that they are so with rats ; and they may be propagated by pigs which eat offal of other pigs containing intestinal trichinæ and embryos, or by their eating the flesh itself, as sometimes happens.]

ADDITIONS TO THE REVISED EDITION OF 1880.

SECTION I.—ACUTE INFECTIOUS DISEASES.

1.—P. 586.

Bacteria have been found in the blood of scarlatina patients, as in that of measles and small-pox patients ; when rabbits were inoculated with this blood, the bacteria were found in their blood. Dr. *Carpenter* thinks that scarlatina is sometimes caused by decomposing blood from slaughter-houses. It has been variously stated that cases occur from defective sewerage. The sources of contagion are so varied and often so obscure that in a large city at least it is difficult to exclude this mode of origin from any case.

Among the complications of scarlatina, acute nephritis is probably the most common. Its symptoms usually begin in the desquamative stage during the third week. In this stage also comes a rarer complication, viz., acute articular rheumatism ; this has the same symptoms and endocardial complications as simple rheuma-

tism, but it is more apt to be accompanied by severe joint-disease and suppuration. Besides these, there may be inflammations of serous membranes, pleuritis, pericarditis, or meningitis, also pneumonia, etc.

The prognosis is less favorable than in measles; mild epidemics are less frequent, and even mild cases often become complicated; this is justly called a treacherous disease. In not a few epidemics the mortality will reach one-third. Cases in children under five years or in old persons, those with high fever, intense and long-continued eruption, or with any of the severe complications, are unfavorable.

2.—P. 607.

Some persons are so susceptible to the poison that it acts even when very dilute, as coming through the windows from the next house, or by letters, paper money, etc. The nature of the poison is not understood. Minute spherical bodies (bacteria) have been found in the contents of the pustule, in the skin around it, and in the internal organs; but it is not certain that these bacteria are the vehicles of the contagion. The results of the experiments of *Chauveau*, *Burdon-Sanderson*, and others, in removing the bacteria from vaccine lymph, do not agree with those of *M. Wolff* and *Hillier*.

Weigert believes that the first changes, which occur in the cells of the rete Malpighii, are not of inflammatory nature, but are analogous to changes caused by diphtheritis in the epithelium. The cells of the rete are transformed into translucent flakes of irregular shape and size, and without nuclei; they cause an irritation, which induces an effusion of lymph that separates the affected cells, forming small interspaces in a sort of mesh-work made by the remains of the epidermis. About these interspaces there is active cell-proliferation, and numerous pus-corpuscles escape from the vascular loops lying under the swollen papillæ of the skin, which gradually fill the meshes as the efflorescence ripens. In regard to the central excavation in many pocks, it is not a constant appearance, and is said by *Rindfleisch* to occur where the pustules develop around a hair-follicle or sweat-gland, and to be caused by the covering of the pustule being held down by the epidermis projecting into these depressions.

On the mucous membranes variola is often accompanied by catarrhal redness and swelling; but where these membranes are exposed to the air, there is a pustular eruption like that on the skin. When patients die in the eruptive stage, the spleen is usually much enlarged.

When delirium occurs in small-pox, it may be due to the fever,

and partly perhaps to the inflammation of the scalp causing hyperæmia of the brain. Delirium is more apt to occur in those who have previously been drinkers. A stage of great excitement is often followed by one of weakness with feeble heart-action, which may end fatally.

As the pocks dry up the parts around become pale and less swollen, and the patients are again recognizable; the inflammatory pain gives place to excessive itching, and probably many of the resulting cicatrices may be due to scratching.

Small-pox has numerous complications, such as bronchitis, pneumonia, pleurisy (often with purulent effusion), diseases of the larynx, meningitis, uterine hæmorrhages, abortions, and affections of the eye and ear.

Some observations seem to show that the protection from vaccination does not begin till twelve or thirteen days after the operation; but it should be resorted to even after exposure, in hopes it may at least render the course of the disease milder. Inoculation, which was formerly in vogue, usually induced very light cases, but deaths sometimes occurred.

It is a question how long a patient convalescent from small-pox continues to produce contagion, and how long he should be isolated. He should be isolated till the stage of desquamation is entirely past—say fourteen days after the disease is over.

Dr. *Waters*, in the London "Lancet," says if sun-light be excluded from the room, the disease is arrested at the papular or vesicular stage, and the pain, itching, and smell are less annoying. Constant applications of emulsion of bitter almonds seem to have the same effect.

3.—P. 654.

If the fresh dejections of a typhoid patient spread the contagion, persons immediately about him would be most apt to contract the disease, but this does not seem to be the case; so the stools probably undergo some change which render the germs in them more active. Some authors believe that typhoid may develop spontaneously, others that it is always due to contagion; the poison from the patient reaching some place favorable to its development, and thence again entering the human body.

Most epidemics of typhoid begin after the heat of summer, when the soil is dried to the greatest depth; but others occur independently of the time of year or of the dryness. Their extent is usually limited, perhaps to a town or one part of a city, or perhaps to a single house, in which case it must be referred to infected drinking-water

or defective drainage. Typhoid is relatively rare during pregnancy, but doubts are rising against the old view that it hardly ever occurs in the puerperal state.

The period of incubation probably varies, but seems to average about fourteen days.

The parenchymatous degeneration of the muscles of the heart may result in passive dilatation ; the feeble action following the degeneration may induce disturbances of the circulation, especially emboli, particularly if the weakened contractions favor coagulations in the heart, and particles from these coagula pass from the right heart into the lungs or from the left into the aorta, and reach the spleen, kidneys, etc., causing hæmorrhagic infarctions and possibly abscesses. Venous thrombosis, especially of the crural vein, not unfrequently follows the impairment of circulation.

Among the nervous symptoms, besides the febrile delirium or stupor, there may be others referrible to cerebral œdema or hæmorrhage, meningitis or embolism ; mental disturbances, which usually recover ; paralyses of the facial, ocular, or vocal muscles, or paraplegia. If these paralyses cannot be attributed to severe changes in the nervous centres, they usually recover.

Besides the albuminuria, which disappears with the disease, there are sometimes hæmorrhagic infarctions or even diffuse nephritis.

In severe cases of typhoid, bed-sores are not uncommon in spite of the greatest care, and as a sequel of the disease there may be numerous furuncles.

According to *Liebermeister*, from 7 P. M. to 8 A. M. is the best time for baths ; this being the natural period for remission, if the patient can be kept for several hours at a temperature approaching normal, he may better stand a brief increase of temperature. Another good time for the bath is about noon, at which time also most patients show a tendency to remission.

Menstruation, which usually comes in typhoid at its regular time, is customarily regarded as a contra-indication to baths ; but in severe cases they may be continued, as they may with almost any of the complications, such as diarrhœa, meteorism, etc. Even in threatening paralysis of the heart, some stimulant may be given, and followed by short tepid baths.

SECTION II.

CHRONIC INFECTIOUS DISEASES.

CHAPTER I.

SYPHILIS.

Of late years the study of syphilis has undergone a complete revolution, and the new doctrines have been adopted with remarkable readiness by almost all prominent writers upon the subject, even by those who a few years ago were their most zealous opponents. In previous editions of this work I have expressed a disbelief in the ancient views, according to which inoculation by one and the same poison at one time acts locally, and at another induces infection and disorder of the entire system; and I there declared my preference for the modern theory, according to which there are two poisons, one of which merely induces local disease, namely, an ulcer at the point of inoculation, accompanied in some instances by inflammation and suppuration of the neighboring lymphatic glands; while the other always gives rise to constitutional disorder, with extensive derangement of nutrition. At that time, however, I did not declare myself so unreservedly in favor of the doctrine of the duplicity of the virus (or duality, to use another common expression) as I now do.

I think that it will be to the advantage of my readers if I devote a few lines to a brief analysis of the belief which a short time ago was universally current, regarding the relation of the chancre, or primary syphilitic ulcer, to constitutional syphilis, or lues venerea. It was supposed that the secretion of the ulcer possessed virulent properties, whereby, when brought in contact with an excoriated surface, or upon penetrating through a thin coating of epidermis, it induced a specific dermatitis, and a *primary syphilitic ulcer*. At this stage, it was believed that the noxious power of the virus often became exhausted; and that, if we could succeed in destroying or in healing the ulcer before the infection from it could involve the constitution, the disease

would remain local; if the attempt failed, then a series of affections of remote parts of the body, resulting from constitutional implication, followed the local ulcer, the secondary and tertiary syphilitic diseases. The formation of an induration upon the base or edges of the primary sore was considered an ominous sign, proving the almost certain infection of the system. Hence the attempt was usually made to destroy recent non-indurated ulcers with strong caustics, while older and indurated sores were nearly always treated constitutionally, especially with mercury, which was almost universally regarded as the antidote for syphilis. Finally, the majority of syphilologists, following the teaching of *Ricord*, supposed that syphilis could only be propagated by the primary sore, or chancre, or, in other words, that the secretion of the chancre was the only vehicle of contagion; and that it was not contained in the discharge of secondary affections, nor in the blood, or secretions of syphilitic persons, a theory which was never fully credited by practising physicians. It would take too long to relate in detail how each of these propositions in turn first was doubted, then was refuted, until at last the whole theory was abandoned as false, or, at all events, as based upon erroneous observation. We shall touch upon some of these errors in laying down the tenets of the modern doctrine.

In the present chapter we shall treat first of the *chancre*, and of the *glandular chancre* or *acute bubo;* after which we shall describe constitutional syphilis, the first manifestation of which consists in syphilitic induration and the primary syphilitic ulcer. To the latter we shall not apply the term chancre, according to the usage of most modern authors.

A.—THE CHANCRE.

As the chancre is not a constitutional disease, it might, perhaps, have been entirely separated from the subject of syphilis, and classified with gonorrhœa, and other affections of the genitals; but, although such an arrangement would be more systematic, yet, considering the extremely frequent coexistence of the chancre with syphilitic induration, it does not seem so practical.

The chancre often is called the "soft chancre," in contradistinction to the sore of primary syphilis, the "hard chancre." Other authors call it the "chancroid," reserving the term chancre for the primary syphilitic ulcer. Other syphilologues apply the epithet "virulent" to the chancre, and call the ulcer of primary syphilis the "infectious" ulcer. It is very desirable that authors should agree as to the names of the two diseases, in order to allay the confusion arising from a diversity of nomenclature.

Etiology.—We shall not discuss the question as to the origin of

the chancrous virus, since we have no means of deciding it; but there is no doubt that at the present time it never develops spontaneously, and that no one nowadays contracts a chancre who has not been infected with its virus. The chancre, then, belongs to the class of purely contagious diseases; and we are fully entitled to employ the term chancre-contagion instead of chancre-virus.

Unlike the contagion of measles, scarlatina, and small-pox, the contagion of the chancre is not volatile, nor capable of poisoning the atmosphere about the patient, and of infecting individuals within its reach. It is of a fixed nature, and is only found in the secretion of the sore, and in the contents of a chancrous bubo. The poison itself cannot be detected in either of these vehicles, either under the microscope or by means of chemical examination. The matter which covers the sore, and the contents of a glandular chancre, do not differ perceptibly, either morphologically or chemically, from the discharge of an ulcer of any other kind, or from the pus of other suppurating glands.

The predisposing conditions for the chancre are usually more general perhaps than those of any other disease. Neither age, sex, nor constitution, seems capable of modifying the susceptibility to its infection. It is true that individuals in the prime of life are more often affected than children or old persons; that men suffer oftener than women, the vigorous and healthy more frequently than the feeble and sickly; but this is simply because the former expose themselves to contagion more than the latter, and not because they are more susceptible to its influence. Hence persons with a thin epidermis are more liable to infection, because slighter injuries suffice to produce solution of continuity of their skin, thereby enabling the poison to act upon the corium. The results of syphilization, that is, the artificial production of chancres by inoculation, seem to show that such inoculations, when repeated a great many times, have the effect of extinguishing the susceptibility to contagion.

By far the most common mode of transmission of the chancre is by coitus with a person who is already diseased. Infection also sometimes results from lewd embraces, from kisses, and from the use of water-closets, tobacco-pipes, tumblers, and similar articles, impregnated with the chancre-virus. Physicians and midwives are sometimes infected in making vaginal examinations, and conversely, women are now and then inoculated by nurse or doctor; but these and all other modes of origin of the chancre, often as they are assigned by patients, are extremely rare in comparison with that of impure intercourse. Excoriation of the cuticle or epithelium, at the point where the poison comes in contact with the genitals, favors transmission of the disease, but it has not been proved that an abrasion of the surface is essential

to infection. On the contrary, chancres are often observed to develop upon parts of the genitals where the most careful search immediately after coitus had failed to detect any breach of continuity of the epidermis. Upon other parts of the body, too, where the epidermis is thin, such as the lips or the nipples, infection may occur without wound of the cuticle, while upon the hand or other region thickly covered with cuticle, as long as there is no breach of continuity, the contact of the poison produces no effect. The seat of the chancre, therefore, is most generally upon the genitals, far more rarely upon the anus, between the breasts, in the mouth, or upon the hands.

Symptoms and Course.—According to the concurrent belief of all trustworthy observers, the period of incubation of the chancre-virus is a very brief one. *Ricord* goes so far as to declare that it has no incubative period, but that changes arise at the point of contact of the poison immediately upon its implantation, although they are not commonly noticed at first, owing to their apparent insignificance.

Course of an Inoculated Chancre.—Upon introducing some of the secretion of a chancre beneath the epidermis, through a puncture from a lancet-point, no change takes place at the point of inoculation during the first twenty-four hours. At about the thirty-sixth hour a slight redness appears, and in forty-eight hours there is a distinct, bright-red macula. In the course of the third day the macula rises into a flattish papule, and upon the fourth day the epidermis is raised, forming a vesicle, surrounded by a reddened areola. In the next day or two its contents become more yellow and purulent, the vesicle transforming into a pustule, which bursts between the fifth and eighth day; or else, together with its contents, dries up into a scab. After rupture of the pustule, or after removal of the scab, we see an ulcer of the size of a pin's head, or perhaps as large as a pea; it is almost circular, and penetrates into the corium in a manner disproportionate to its size. Its edge, which is œdematous from inflammation, has a puckered appearance. In the next few days, the base and edges of the sore are attacked, by a diphtheritic process, and it begins to enlarge. As the elements of the tissues are necrosed, and broken down into detritus, the base of the sore assumes a grayish, dirty, lardaceous appearance. As the diphtheritic destruction goes on irregularly at its periphery, the borders of the ulcer acquire a gnawed, ragged form. If the point of inoculation be destroyed by caustic within the first four days, the destructive process may generally be cut short. After the fourth day this is scarcely ever possible.

A chancre of accidental origin begins either by a macule or papule, upon which, after repeated exfoliation of the cuticle, an excoriation and loss of substance takes place, or else a vesicle or pustule forms

first and bursts; or, what is more common still, the chancre proceeds from infection of a laceration, which, instead of healing immediately, becomes covered with a dirty-looking exudation. The characteristics of a sore arising in this way are not always so well marked as to admit of its ready distinction from other forms of ulceration. In doubtful cases, therefore, it is very advisable to inoculate the thigh of the patient with some of the secretion of the sore; two or three such punctures should be made, and should be covered with a watch-glass attached by adhesive plaster. As we shall hereafter learn, the inoculation of a patient having a syphilitic ulcer with some of his own matter results negatively. When the inoculation takes place, and the changes described above occur, there can be no further doubt of the chancrous nature of the ulceration. For the present, we shall merely describe the more common forms of simple uncomplicated chancre; then, after giving an account of the primary syphilitic ulcers, we propose to treat of the modifications of the chancre which arise from its complication with syphilitic induration.

The *common diphtheritic chancre* is characterized by ragged and detached edges. The loss of substance looks as if it were made by a punch. Its most common seat in men is upon the inner surface of the prepuce, the surface of the glans and sulcus between the prepuce and glans, and, above all, the frænulum; less frequently, it is upon the outer surface of the prepuce, or the integument of the penis. Ulcers of the frænulum are nearly always deep and excavated, and the frænulum itself is perforated. When the sore is situated in the sulcus, between the prepuce and glans, the ulceration is apt to extend, by spontaneous inoculation, over a large portion of the corona glandis. If it penetrate deeply at this point, the loose subcutaneous tissue becomes infected by the secretion, and gives rise to a virulent abscess. When seated upon the glans, it usually penetrates more profoundly than when upon the prepuce, although perforation down to the urethra, with the formation of a urethral fistula, is rare. The urethral chancre is not common. It usually begins at the mouth of the urethra, and a small loss of substance which extends inward is visible upon its swollen and deeply-reddened lips. At other times the urethral chancre is more deeply situated, and only betrays its existence by the purulent flow from the meatus and by pain in the urethra, situated at some particular point, and which is increased by pressure or by making water. If a concealed urethral chancre be not complicated with gonorrhœa, it is easy to recognize it, as the scantiness of the purulent flow will attract attention, and prevent its being mistaken for a clap. Successful inoculation, however, is the only means of making the diagnosis sure. It is hardly ever detected, though, when accompanied by a gonorrhœa.

as, owing to the copiousness of the discharge, and to the absence of other conspicuous symptoms, inoculation is almost always neglected In women, the most frequent seat of the common chancre is the vulva upon the posterior commissure, and at the entrance to the vagina. In very rare instances it appears in the vagina, and even upon the vaginal portion of the neck of the womb. When the common chancre begins to heal, the destruction ceases, and the lardaceous appearance of the base of the ulcer disappears ("the chancre cleans up"), granulations appear upon the base and edges of the ulcer, which gradually fill up the loss of substance. After the chancre has healed, there remains a cicatrix, more or less distinctly stellated, according to the depth of the ulcer. The period at which the process of repair commences is very variable. Some chancres become covered with granulations, and transform into simple sores, which do not secrete inoculable pus in a week or two, while others go on extending for months, and retain all their characteristics; that is, ragged edges, lardaceous base, and virulent discharge.

The *superficial chancre* occurs most frequently upon the glans and prepuce in men; and between the labia and nymphæ, and at the entrance to the vagina, and on the neck of the womb, in women; and it appears in both sexes with equal frequence on the skin. When situated upon the glans, an exact counterpart of the ulcer is usually found upon the corresponding inner surface of the prepuce. The form of the ulcer is irregular, and its surface looks as if the cuticle had been scalded off. At its edge there is a white border. The destructive process never extends deeply. When situated at the orifice of the prepuce, cracks occur in its folds, which render retraction of the prepuce very painful. The superficial chancre often results in phimosis and paraphimosis. In the former instance it is almost indistinguishable from balanitis, excepting by means of inoculation. When a superficial chancre is situated upon the external integument, the scanty secretion soon dries up into a thin crust, and it is not until after it has been treated with a wet dressing that its raw, reddish-yellow surface, sparingly covered with secretion, becomes visible.

The *follicular chancre* develops from a sebaceous gland. Its surface is very small, disproportionately deep, and long retains its regular round form.

The *phagedenic chancre* usually originates from the common chancre. Its secretion is thin, ichorous, and very offensive; its form is irregular, its base is of a grayish-white or greenish color, consisting of necrosed tissue and of infiltration; its edges are livid, and surrounded by coppery-red areola. It spreads rapidly, and in men it sometimes destroys a large portion of the prepuce, glans, skin of the

penis, and scrotum; and in women, eats away the labia, perinæum, and parts about the anus. The phagedenic ulcer only appears in broken-down, cachectic subjects, and loses its malignant character as the constitution improves. Then the devastation ceases; the diphtheritic coating upon the base of the ulcer is cast off; healthy granulations form, and the malignant, eating sore changes into a simple ulcer, with a tendency to cicatrize. If the constitution of the patient does not improve, or if it becomes still more depraved by the further exposure to noxious influences, especially by the use of mercury, the destructive process, which continues without stopping, finally becomes complicated by a slow fever which consumes the patient.

The *gangrenous chancre* is sometimes a modification of the phagedenic chancre, while at other times it develops from the ordinary form of the disease; in either case, the base of the ulcer and the parts about it become bluish, and are afterward converted into a black, insensible moist slough. There is a dusky redness immediately around the eschar, which is encircled by a somewhat extensive and severe œdema. The disease may advance until it has destroyed a large portion of the penis, or labia and perinæum, and life itself may be imperilled by the constitutional disturbance which accompanies the gangrene. At other times the destructive process ceases sooner; a line of demarcation forms, the sloughs separate, and the disease recovers, leaving a more or less extensive loss of substance behind it. We do not always know why chancres become gangrenous. Now and then, owing to the action of unknown causes, gangrenous chancres become of very frequent occurrence, or, at least, are more common than usual. In many instances, however, mechanical action, such as straining or tension of the inflamed part (as, when the chancre is complicated with phimosis or paraphimosis), chemical agents, or irritation of the sore by the putrefaction of retained secretion, may be regarded as causes of the malady.

Treatment.—In proportion as the opinion has gained ground that the chancre never leads to constitutional syphilis, the old and erroneous practice of treating chancres by mercury has died out. The progress of knowledge has speedily borne fruit in this instance, the importance of which cannot be sufficiently prized. We have only to consider that, but a few years ago, at least one-half of the physicians used to subject all patients with chancres to a course of mercury, and thus often enough ruined their health by means of this pernicious poison, under the mistaken impression that they thereby averted induration of the ulcer, and prevented constitutional contamination. Nowadays, a physician who treats a simple chancre with mercury makes a gross blunder. On the other hand, I do not think it judicious to make the treatment of a simple chancre a purely local one. It will heal much more rapidly if the

patient be brought under favorable hygienic influences, and if he be defended from all noxious agencies throughout the course of the disease. If the circumstances permit, it is well to confine him to his chamber, or, at all events, to forbid all unnecessary walking. Moreover, since the majority of this class of patients are young and vigorous, living more or less freely, and drinking spirituous liquors, it is generally advisable to place them upon a rigid diet (gruel morning and evening, with broth and a little meat at mid-day), to forbid coffee, beer, wine, or spirits, and now and then to give small doses of some saline cathartic (one or two glasses of Friedrichshaller, or Püllnaer bitter water), which may be taken in the morning, fasting. Although a chancre usually heals much more quickly under this treatment than when a strict regulation of the diet is neglected, it is merely because of the luxurious mode of life led by most patients who have exposed themselves to the infection of the chancre. The above directions are not applicable, and indeed might do great mischief, and retard the healing of the sore, if applied in the case of a badly-nourished, cachectic patient. In such a case, the diet must be nutritious and rich; the use of wine and beer is indicated, and often exerts a favorable effect upon the healing of the ulcer.

The local treatment of a chancre must be similar to that of any other atonic ulcer, of which the chancre may be regarded as the prototype. Until after the fourth or seventh day, it is advisable to destroy the ulcer by caustics. For this purpose, it is best to make use of the potassa fusa, the Vienna paste (quicklime, five parts; caustic potash, six parts), or the chloride of zinc, which makes a dry eschar (℞ zinci chlor., butyr. antim., āā ʒ ij, f. c. pulv. alth. past. mollis). After the seventh day it is not advisable to cauterize, as then the process of healing is retarded rather than promoted thereby. As a rule, ointments should not be used, and the sore should be dressed with warm camomile-tea, or other mild, stimulating liquid. The most common applications are the aromatic wine, the black-wash (calomel ʒ ss; aquæ calcis ℥ ij), the yellow-wash (hyd. chlor. corrosiv. gr. j; aquæ calcis ℥ ij). The blue-wash (cupri sulp. gr. j; aquæ ℥ j), which is almost exclusively used by *Von Bärensprung*, is also highly to be recommended. When the chancre is very painful, and when its discharge is very profuse, compresses, wet with lead-water and cold sitz-baths, are of benefit. It is generally sufficient to dress the wound twice a day, after previously washing it or bathing the part in camomile-tea. Too frequent dressing does harm. If, in spite of this treatment, the floor of the ulcer retains its lardaceous aspect, it is advisable to sprinkle the surface now and then with a thin layer of red precipitate. After the sore has lost its specific character, and nevertheless is slow in healing, it may be touched lightly with lunar caustic, or dressed

with a wash of zinc or lead. When the frænulum has been perforated, it is best to divide the remaining bridge of mucous membrane at once. Where a phimosis prevents the proper treatment of the chancre, injections beneath the prepuce must be practised methodically, in order to remove the accumulated secretion. It is sometimes necessary to operate for phimosis; but this is never to be done excepting in urgent cases, as the entire wound generally becomes converted into a chancre.

In the treatment of the phagedenic chancre, the general health of the patient must receive our first attention. His strength is to be husbanded rather than reduced; and a nourishing diet, and sometimes wine, bark, and iron, are indicated. The utmost cleanliness is to be observed. As a topical application we recommend dressings of dilute solution of acetate of lead, or of chloride of lime (℥ ss— ℥ j to water ℥ vj).

Treatment of the gangrenous chancre is to be conducted upon similar principles, paying due attention to the surgical rules for the treatment of mortification.

APPENDIX.

THE GLANDULAR CHANCRE.—THE VIRULENT, ACUTE BUBO.

Etiology.—We have seen that the virus which the chancrous matter undoubtedly contains (although we are unable to isolate it or to demonstrate it to view) may be transferred to other parts of the body, and that it will cause inflammation and ulceration at its point of implantation. In a similar manner the virus is very often taken up by the lymphatic vessels, and conducted to the neighboring lymphatic glands, in which it also gives rise to specific inflammation and suppuration. A lymphatic gland thus inflamed and suppurating from absorption of the chancre-poison is called an acute, virulent bubo, or, what is better, a glandular chancre. All chancres do not cause virulent adenitis with equal frequency. It is most apt to accompany an ulcer of the frænulum. Neglect of cleanliness and a too irritating mode of treatment seem to favor absorption of the virus, and the production of the virulent bubo. Cases seem to occur now and then, in which the virus reaches the corium through the epidermis without producing any lesion at its point of entry, but is there taken up by the lymphatics, carried by them to their glands, where it gives rise to specific inflammation and suppuration. Such a bubo, which has not been preceded by a chancre, is called a "bubon d'emblée."

Symptoms and Course.—A virulent bubo generally makes its appearance in the second, third, or fourth week, more rarely in the fifth or sixth week, and it sometimes develops immediately after the forma-

tion of the chancre. The first symptom of this troublesome complication consists in a feeling of pain at a circumscribed point in the groin, not far from the genitals. Upon examination, we find a small lump, which, already even, is sensitive to the slightest pressure, and which corresponds to a moderately enlarged inguinal gland. The disproportion between the severity of the pain and the smallness of the swelling, the proximity of the latter to the genitals, the fact that one or two only of the glands are enlarged and never a large number of them, make us aware, even thus early, that we have to deal with an acute virulent bubo, and not with the so-called sympathetic bubo, or with the syphilitic glandular enlargements hereafter to be described. The further course of the virulent bubo varies. In very rare instances the inflammation is resolved, and the pain and swelling of the affected gland gradually subside. In such cases it is not improbable that it is merely the inflammation which has spread along the lymphatic vessels to the gland, but that the chancrous virus has not been absorbed with it, or, in other words, that the bubo is a sympathetic one. The process above alluded to, namely, the extension of an inflammation along the lymphatics to the lymphatic glands is a very common occurrence also among non-specific ulcers. Far more frequently, and probably always, in the case of a virulent bubo, the inflamed gland suppurates. In favorable cases the pus soon perforates the capsule of the gland and the adherent skin which covers it. In other instances, the connective tissue about the gland becomes the seat of an extensive inflammatory exudation, the tumor becomes much enlarged, and is no longer movable. It becomes very difficult for the patient to walk, and, in spite of the efforts which he usually makes to conceal it, we can perceive that he favors the affected side as he steps. In persons with a tendency to fever, there is also an elevation of temperature, acceleration of the pulse, general *malaise*, and other febrile symptoms. Considerable time elapses thus, ere the skin begins to redden, and ere the circumscribed point of fluctuation makes its appearance. If a puncture then be made at this point, or if the skin assume a dark-red hue and grow thinner and thinner, until the matter is discharged spontaneously, a small portion only of the swelling subsides, and it is often as large as a man's fist, and of an irregular, knobby form. By-and-by fluctuation, followed by escape of matter, occurs at other points; but, in spite of the numerous abscesses, weeks and months often pass by without any diminution in the size of the tumor in which they form. The course of a virulent bubo is equally tedious and intractable, if the matter which has escaped from the inflamed gland into the surrounding areolar tissue have formed sinuses and fistulous tracts, as well as when the accumulation of matter within the gland is slow to point and

does not discharge itself until the inflammation of the surrounding tissues has also resulted in numerous abscesses. After breaking of the abscess and discharge of the matter, the resulting ulcer shows the properties of a chancre and discharges inoculable pus. Its edges are ragged, bluish red, and generally somewhat undermined; its floor is covered with false membrane, and it is very slow to heal. A chancre thus produced from a virulent bubo may become phagedenic or gangrenous, and may give rise to wide-spread destruction, which sometimes terminates in erosion of the femoral vessels, or in peritonitis.

Treatment.—It matters little whether a virulent adenitis be accompanied by a chancre or not; in either case, the probability that the inflammation of the gland will not end in suppuration is so small, that we may spare the patient the applications usually recommended to discuss a bubo, such as leeching, inunction of blue ointment, methodical bandaging, and the like. I usually cover a bubo with a simple plaster, generally the emplastrum fuscum (emplas. galbani. co.), and make it fast with a spica bandage. Under such treatment the bubo now and then subsides; in other instances, it suppurates without causing the patient much annoyance. Should fluctuation appear at a point which unmistakably corresponds to the gland, I immediately let out the matter through one or more punctures; but if the surrounding areolar tissue be already in a state of phlegmonous inflammation, and if fluctuation appear, which does not seem to be in a gland, but to proceed from a phlegmonous abscess, I am not so hasty in opening it, but prefer to wait until the parts about the softened spot are also in a state of suppuration, and until the hardness has disappeared under the pressure of the pus. As soon as this occurs, and after the skin over the point of fluctuation has become well thinned, I convert the entire top of the abscess into an eschar, by persistent rubbing with caustic potash. During this process (which I learned at the Hamburg hospital) the adjacent parts must be protected from contact with the liquefying potash. This treatment is, no doubt, painful, but has this great advantage, that no fistulous track nor sinuses ever form, and that, the day after the separation of the eschar, the bottom of the abscess presents the aspect of a healthy ulcer. According to my experience, also, this cure is much more rapid than when we have to make puncture after puncture, as fresh points of fluctuation show themselves, to lay open fistulæ as they form, and to remove the undermined edges of the ulcer.

Latterly, I have often had recourse to another mode of treating virulent buboes, and with striking effect. I put a vesicatory upon the tumor, cautiously open the resulting blister, and then allow a new cuticle to form under a simple dressing. I then immediately blister

again, and repeat the procedure until the infiltration about the gland, which usually begins to resolve after the first vesication, disappears entirely. Then, if a fluctuating point appear, I puncture it, or destroy its covering with caustic paste. In none of the cases thus treated by me have any sinuses or fistulæ formed

B.—CONSTITUTIONAL SYPHILIS.

The question as to the origin of the syphilitic poison is quite as obscure as is that of the origin of the chancre-virus. At the present day, syphilis is a purely contagious disorder. Its venom reproduces itself in the organism which it infects, and the transplantation to another person, of the virus thus reproduced, is the only manner in which syphilis can be propagated.

The exact nature of the syphilitic virus, or, as we may say with equal propriety, the syphilitic contagion, is unknown, since we are unable to obtain it in an isolated form, and to test its chemical and physical properties. Like the chancre-virus, however, it is of a fixed character, and does not pervade the atmosphere around the patient; but its habitat is by no means confined to the secretion from the syphilitic ulcer, and to the contents of syphilitic tumors, for it is also contained in the blood. It does not seem to exist in the natural secretions of the body, such as the saliva or urine, nor in pathological exudations produced by intercurrent disease. Thus, if we inoculate a healthy child with pure vaccine lymph, obtained from a syphilitic subject, the child thus vaccinated does not become syphilitic; but, if the lymph contain a little blood, which is a vehicle for the syphilitic poison, the latter will also be implanted upon the patient. It has not yet been determined whether the virus exists in the milk of a syphilitic woman. The frequence with which the disease is transmitted to infants, from wet-nurses, may be due to the existence of bleeding excoriations upon the nipples of the nurse. In the following chapter we shall treat of its propagation during the act of generation.

Liability to syphilis is so general, that an immunity to its virus, if it exist at all, is certainly very rare. The fact that healthy persons, adults, and men, are more frequently infected than invalids, women, or children, is simply because the former are more apt to be exposed to the disease than the latter. Like the chancre-virus, the syphilitic poison is more difficult of inoculation when the cuticle is thick and resisting than when it is the reverse, a circumstance which argues in favor of the practice of circumcision. Infection is also more likely to occur when the genitals are strongly developed than when they are small. Eczema of the glans and prepuce, likewise, greatly increases the danger. Notwithstanding this very general susceptibility to the syphilitic

virus, however, the system once contaminated by it obtains an immunity almost absolute against subsequent infection. This very interesting fact is analogous to the freedom from further attacks of scarlatina, measles, and small-pox, enjoyed by persons who have once suffered from those diseases. If we inoculate the discharge of a syphilitic sore, or any other vehicle of syphilitic contagion, upon a person who has a primary syphilitic induration or syphilitic ulcer, at the moment of inoculation, or who has already had one, we shall obtain negative results. It is this fact which has given rise to such gross errors. For a long time experimental inoculation was only practised upon individuals who were already syphilitic, and, from the negative results which followed, when blood or the secretion of secondary sores was employed, it was inferred that blood and secondary secretions were not contagious. It was not until it was determined to inoculate healthy, or, at least, non-syphilitic persons with these vehicles that it was ascertained that the want of result of the former experiment was simply due to the immunity of syphilitic persons to further infection, and that their blood, and the discharge from secondary syphilitic ulcers, were quite as infectious as the secretion of a primary sore. Exceptions to this rule seemed to occur; inoculation of the secretion of a primary sore upon the person having the sore resulting sometimes in another sore. It is now determined that in such cases the syphilitic ulcer is complicated by the chancre. The ulcer which follows the inoculation is a chancre, and not a syphilitic ulcer. Contamination of the system with syphilitic virus affords no protection against infection by the chancre, any more than does the preëxistence of one or several chancres secure the patient from further action of the chancrous virus.

Since even a person having a primary syphilitic ulcer or induration, but who has no secondary symptoms, is already secure from further syphilitic infection, we may regard the primary induration and ulcer as a sign of contamination of the system, and as the first symptom of constitutional syphilis. Since, however, the above-named affections stand first in the series of disorders which occur, and as the first sign of constitutional infection always appears in the form of an ulcer or induration at the point of entry of the virus, the terms "primary induration" and "primary sore" may be regarded as perfectly appropriate. The immunity against fresh infection, which contamination with syphilitic poison affords, accounts for the interesting and formerly very perplexing fact that infants are infected by syphilitic nurses more frequently than by syphilitic mothers. Generally, a child sucking at the breast of its nurse is not at first syphilitic, and, therefore, is liable to infection, while the child which sucks the breast of a syphilitic mother is nearly always syphilitic itself, and hence can not be inoculated.

By far the most frequent exciting cause of syphilis is coitus with an infected individual; but there are many well-authenticated cases where infection has been conveyed by the hand of a physician or midwife, by using a pipe, or tumbler, or privy, polluted by the venereal virus. There is no doubt, moreover, that the disease has often been propagated by vaccination, and another series of cases is known to have sprung from the use of dirty lancets, and in bleeding and cupping, by careless surgeons.

Symptoms and Course.—I. *The Primary Syphilitic Induration, and Primary Syphilitic Ulcer.*—The period of incubation of the syphilitic virus is from three to four weeks. A correct knowledge of this fact is of recent date. Prior to this discovery, which was made by inoculating healthy subjects with syphilitic virus, we were unable to account for the phenomenon that, in a large number of cases, a chancre (chancroid), after lasting for some weeks, would become indurated at its base and edges; and this it was which caused the false impression that a soft chancre could assume a pernicious character, and change into a hard one. Experience has taught that the indurated chancre is almost always the precursor of secondary symptoms. The following is the correct interpretation of these facts: Both poisons, the chancrous, as well as the syphilitic, act simultaneously upon one and the same point; in a few days the chancre forms; the period of incubation of its virus being a very short one. Three or four weeks afterward, the time of incubation of the syphilitic contagion having expired, the syphilitic induration develops at the base and edges of the ulcer. It may happen that the chancre has healed before the incubative stage of the syphilis has terminated. In such a case an induration forms in the scar of the chancre. The conversion of a syphilitic induration into a chancre, by the implantation of chancrous virus, may also take place.

[Some observations seem to indicate that the poison of chancre and chancroid may be present on the same sore, giving a "mixed ulcer;" so that an apparently soft chancre may become indurated and be followed by constitutional symptoms, and, on the other hand, inoculation from an apparently hard chancre on the bearer of it may induce a chancroid. It has, however, been asserted that inoculation of pus of *any kind* in a syphilitic patient may induce an ulcer resembling a chancre. Even on this point, where so many observations have been made, opinions are not undisputed; and we may consider that the relation of chancre and chancroid to each other, and to syphilis, is not absolutely determined in all details.

After exposure to the disease, some time elapses before the development of the indurated sore, then some weeks longer before the

appearance of the other symptoms of constitutional syphilis. There is considerable regularity in the succession of these symptoms, reminding us of the course of the acute exanthemata. There may even be premonitory fever and enlargement of the spleen. The so-called "secondary" symptoms affect the skin and mucous membranes; in the "tertiary" stage there are changes in the subcutaneous and submucous connective tissue, the bones, fibrous and serous membranes, and internal organs. This division into stages is not always accurate, as they may extend into each other; but usually they are separated by an interval of latency more or less long.

Chancroid sometimes occurs in the anus and rectum from unnatural coitus, or from the pus of a neighboring chancre. A careless physician may mistake syphilitic ulcerations of the anus for hæmorrhoids; they may cause stricture of the rectum. If syphilitic iritis be accompanied by much dimness of vision, this may be due to cloudiness of the vitreous or to retinitis.

When the periosteum and bones are diseased, the joints may become affected from the same cause; but other joint-affections may occur incidentally, and be called syphilitic rheumatism. Even in these cases, iodide of potassium is very useful.

All authors do not agree that treatment of the primary sore is useless; but some claim that where it is recent and favorably situated (on the prepuce, etc.), it may be excised and sequelæ be thus prevented. *Fournier* says, in treating syphilis, he gives one or two grains of iodide of mercury daily for two months, then discontinues for one month; a second treatment for two months is followed by an interval of three months, and a third treatment for one or two months. This treatment with increasing intervals is continued for two years. The second year iodide of potash is combined with the mercurial.]

After inoculation of a suitable subject with syphilitic virus, whether accidental or intentional, that is to say, of a subject who is not syphilitic, and who has never been so, and after expiration of the term of incubation, there arises, not a vesicle nor pustule which breaks, leaving a sore—as occurs after infection from the chancre—but a hard papule, or nodule of variable size and thickness. The smaller syphilitic indurations are of the size of a lentil or pea, the larger attain the size of a bean, or of a small hazel-nut. Microscopic research shows the induration to be the product of profuse proliferation of cells and nuclei, which have no characteristic peculiarities, and which lie embedded between the normal elements of the parts, in the scanty connective tissue. The epidermis, or epithelium, over this hard infiltration, at first does not exhibit any lesion; but soon the epithelial covering begins to exfoliate,

the new coating becomes thinner, the surface shows a peculiar glittering, dirty redness, and, after repeated exfoliations, the epithelial coat is not renewed, but the surface remains raw, and gives off a scanty secretion. In other instances the surface forms a scab, which, after separation, leaves an ulcer with a hard base. The primary syphilitic ulcer may be called the ulcerative induration of syphilis. Its most common situation is upon the genitals; in men, upon the inner surface of the prepuce, and in the coronary fossa; in women, between the nymphæ, at the posterior commissure, and at the entrance to the vagina. Sometimes, however, it appears upon the nipples, at the angles of the mouth, upon the tongue, and not unfrequently upon the fingers. No part of the body is exempt, and a syphilitic induration will arise wherever the virus touches a spot where the cuticle is thin, or where it has been abraded. The localities above mentioned, however, are the ones most favorable to the occurrence of infection. When but a single follicle is implicated, the induration assumes the form of an upright cylinder, and, if a series of them lying close together be involved, the confluence of these indurations forms a hard elevation. When the induration is situated upon the lips of the meatus urinarius, that orifice forms a somewhat gaping, rigid funnel, which feels cartilaginous to the touch. Not unfrequently, when the mouth of the prepuce is somewhat narrow, its anterior border becomes fissured during coitus. If this point becomes indurated in consequence of syphilitic infection, the prepuce can no longer be drawn back, its outlet having been converted into a hard ring. A phimosis of this kind will subside after the induration has been resolved.

The bottom of a syphilitic ulcer is not lardaceous, its edges are not eroded, nor does it show any tendency to spread. All these characteristics are peculiar to the chancre (chancroid), nor is a syphilitic sore painful, like a chancre, being usually very indolent. It is also solitary, as a rule; and it is only as an exception, when several points are infected simultaneously, that several syphilitic sores are ever seen together. Until the induration ulcerates, it is often overlooked; and it is not until the sore is established, that the attention of the patient or physician is drawn to it. Sometimes, the induration is so slight, that the sore may be mistaken for a chancre. In such cases all doubt may be dispelled by inoculation.

The following are the more important forms recognized among syphilitic ulcers:

The *superficial* ulcer (*Ricord's* chancre parcheminé). In this variety the induration forms a thin cake. An attempt to pinch up a spot of this kind between the fingers imparts a feeling as though a bit of parchment had been slipped beneath the ulcer. Its surface secretes

a scanty thin liquid, in which no pus-cells can be found under the microscope, but only a granular detritus. It often heals quickly, but the secondary symptoms follow this form of sore quite as surely and promptly as any other variety.

The *elevated* sore (ulcus elevatum) presents an excoriation which is almost void of discharge, seated upon an indurated base of varying thickness and consistence, by which it is elevated above the surrounding level. From time to time it is coated by a thin layer of epithelium, which generally soon exfoliates in fine scales, leaving a new excoriation.

The *Hunterian* chancre (ulcus vallatum) not only has a hard base, but is surrounded by an elevated, hard, callous border, so that it is deeper in the middle than at the periphery. It rarely heals in less than five or six weeks.

All the different forms of syphilitic sore may become phagedenic, that is, they may be attacked by a rapidly-extending diphtheritic process. The destruction then often spreads beyond the limit of the induration, largely involving the skin or mucous membrane. When the syphilitic induration is associated with the chancre, the operation of both poisons upon the same point results in a modification of the ulcus vallatum, that is, the border of the chancre becomes hard and callous, and surrounds it like a wall. At a later period the induration likewise appears in the bottom of the ulcer.

The duration of a syphilitic induration or a syphilitic sore varies. Three months, at least, nearly always elapse ere the hardened spot recovers its normal consistence. The induration often lasts half a year, and even longer. It is remarkable that, as the secondary symptoms begin to appear, the induration begins to dissolve, and then soon subsides, leaving behind it a brown pigmented spot. The pigment marks the former site of the disease for a tolerably long time. When it finally disappears, the spot remains whiter than the adjacent parts, like the cicatrix of a neoplastic growth. Unless the indurated spot has also been the seat of a chancre, there is no depression.

II. *The Indolent Bubo and Extended Syphilitic Disease of the Lymphatics.*—An acute enlargement of the lymphatic glands occurs in many of the acute infectious diseases and in syphilis, the prototype of the chronic infectious maladies, the lymphatic glands always participate in the disorder induced by infection of the system by this pernicious poison. The changes which take place in the glands consist in a cellular hyperplasia, and they are but seldom the seat of actual inflammation or suppuration. More frequently, especially when the disease has been of long standing, caseous metamorphosis occurs here and there in them, and this is afterward followed by a calcification. In a few days after its development, the primary syphilitic induration

almost always gives rise to a painless or else to a very slightly painful enlargement of the inguinal glands—to indolent buboes. The number of the glands implicated is always somewhat large, and they are seldom very greatly swollen. Each gland usually attains the size of a bean or almond; and it is exceptional, and only in scrofulous subjects, that it becomes as large as a walnut. The surrounding connective tissue is not affected; the glands remain isolated, and, even when their number and the degree of tumefaction are such as to produce a large lump, it is always of an irregular nodulated shape, and it can readily be perceived that it consists of a conglomeration of isolated glands. Sometimes a thickened and indurated chain of lymphatic vessels can be detected between the primary sore and the indolent bubo. In the rare instances in which indolent buboes suppurate, the skin becomes adherent to the glands beneath it, and gradually reddens, and the tumor becomes painful; some time elapses, however, before fluctuation appears. If, finally, the purulent contents escape, or if they be evacuated artificially, they leave sinuous and tedious fistulous ulcers behind them. Involution of an indolent bubo proceeds very slowly. Several months always elapse ere the swelling of the glands diminishes, and often the last trace of the enlargement does not disappear for years. Whenever cheesy metamorphosis with calcification has occurred, small hard elevations remain for life. The usual seat of primary induration being upon the genitals, indolent buboes are generally found upon the inguinal region; and it is almost without exception the glands which lie above the fascia lata, on the side corresponding to the seat of the primary sore, which are affected. In primary affections of the mouth and fingers, it is respectively the submaxillary and axillary glands which are involved.

About five or six weeks after the formation of the indolent buboes, upon careful investigation, we shall find numerous enlarged lymphatic glands, lying far away from the point of infection and from the indolent bubo, and situated in various regions of the body. This enlargement is most frequent and conspicuous in the cervical and axillary glands, the inguinal glands of the opposite side, and the cubital and submaxillary glands. The size attained by these enlargements varies from that of a pea to that of a bean or hazel-nut. They are quite painless; and, even when pressed upon, are not more sensitive than other parts of the skin. These diffuse glandular swellings often last for years; and, as long as they continue to exist, the syphilis is not extinct, even though no other manifestation of lues be discoverable. Inflammation and suppuration of such glandular swellings are quite exceptional, and are then always the consequence of a complication.

III. *Condylomata.*—The only condylomata which are of syphilitic

origin are the broad ones covered by a thin coat of epidermis, and which, if seated upon the skin, are usually in a state of superficial ulceration, and bathed in a slimy, ill-savored secretion. The dry, pointed condylomata, which are covered by a thick layer of epidermis, are the result of local irritation, and most commonly appear at points moistened by gonorrhœal discharge. The syphilitic condyloma is one of the most frequent symptoms of general infection of the system, and it rarely fails to appear in the series of morbid processes which gradually develop under the influence of the venereal poison. As a rule, too, the condyloma is the first symptom which succeeds the primary affection and the enlargement of the lymphatics. Its external aspect and histological character have already been described. Upon the skin, the most frequent seat of the soft condylomata is between the nates; in women, between the labia; and in men, upon the scrotum and outer surface of the penis. Sometimes they spread over the inner surface of the thighs. Their appearance at the angles of the mouth is quite common; they are more rare at the commissure of the eyelids, between the toes, and under pendulous breasts. They often become fissured, especially when they have coalesced so as to form extensive growths, and in this way painful and obstinate ulcers often form. Fissures and cracks (rhagades) frequently appear in the skin about the condylomata. They are attended by severe pain and heal slowly. We have already treated of the condylomata of the mucous membrane, of the mouth and fauces (plaques muqueuses), and of the condylomatous growths which accompany syphilitic laryngeal ulceration.

IV. *Syphilitic Disease of the Skin—Syphilides—Syphilitic Exanthemata.*—Syphilitic affections of the skin depend partly upon hyperæmia and exudation, and partly upon the development and metamorphoses of the "gummy tumor" (tubercular syphiloma) of the skin. In the former class the exudation is sometimes thrown out upon the surface of the skin, sometimes within the tissues, and sometimes in both together. A classification of the syphilides into many species has been based upon the modifications thus induced, and upon the variety in extent and magnitude of the eruption. Since, however, the classification of all other non-specific hyperæmic and inflammatory cutaneous affections is also founded upon similar data, we may be brief in our discussion of the syphilides, and confine ourselves to a description of the differences by which the syphilitic exanthemata may be distinguished from the non-syphilitic ones.

The general peculiarity of syphilitic eruptions, upon which most weight has always been laid, is their color; and, indeed, all syphilitic exanthemata, when of somewhat long standing, are of a peculiar coppery-red hue; and chronic eruptions which do not present this color,

even though they appear upon notoriously syphilitic subjects, ought not to be regarded as of syphilitic origin. This proposition, however, does not admit of inversion. A rash need not be syphilitic because it has a coppery color, as is sufficiently proved in cases of psoriasis and of acne rosacea (commonly known as "copper nose," on account of its appearance). The origin of this color is to be attributed to the addition to the redness, caused by the capillary hyperæmia, of yellow, blue, and brown pigment, the result of minute extravasations of blood, and the metamorphoses of the hæmatin thus set free. In very recent cases the coppery hue is not observable, no extravasations having as yet occurred, or because the effused blood has not yet undergone change of color.

Another characteristic of the syphilitic exanthemata (which is closely related to the foregoing one, as it also depends upon gradual transformation of the coloring matter of extravasated blood into pigment) is, that, after they have recovered, stains of pigment are almost always left in the skin.

Syphilitic eruptions may be further distinguished from non-syphilitic ones, from their type being still less distinctly marked than that of the latter. In the same subject we almost always find patches which belong in part to one and in part to another species, and here and there a spot about which we shall be in doubt what category to assign it to For instance, there are often red spots which we hesitate to call roseola, because they are covered with scales of epidermis; but, on the other hand, we cannot regard them as psoriasis, because their coat of epidermic scales is so much thinner than in the non-syphilitic psoriasis.

The situation of the efflorescence is also of great importance in distinguishing between the simple and the specific eruptions. The specific exanthemata are most apt to appear upon parts of the skin which are exposed to the air, and upon such as lie immediately above the periosteum. It is a very suspicious circumstance, when a rash which has a peculiar predilection for certain localities—as psoriasis has for the knees and elbows—appears elsewhere, upon some unusual region instead of upon its favorite seat—as when psoriasis affects the palms of the hands. Another peculiarity of a syphilitic eruption is, that the various efflorescences generally take the form of circles, or of segments of circles. It is to be borne in mind, however, that it is only when this circular arrangement is the result of grouping, and not when it proceeds from healing of a disease in the middle while it continues to spread at its edges, that it is characteristic of syphilis. Finally, it may be added, that syphilitic eruptions scarcely ever itch. In order to determine the question of the syphilitic origin of a doubtful case, it is of importance to ascertain whether it has been preceded by a primary

ulcer, and whether there be any other concomitant syphilitic disease. The crusts and scabs which form in syphilis are distinguishable by being much thicker, as a rule, than those of non-syphilitic origin. This is generally because, beneath the dried, purulent contents of a syphilitic pustule, there is usually an ulcer, the product of which also thickens and dries into a scab. The kidney-shape or horse-shoe shape of specific ulcers of the skin is also somewhat characteristic. This is due to the healing of the sore at one edge while it spreads at another.

The most common form of syphilitic cutaneous disease is the maculous exanthema, the roseola syphilitica. This is an eruption of small, irregular round spots of roseola, which here and there are often confluent; and its appearance is often preceded by a febrile disturbance. The favorite seat of the rash is upon the belly, on the sides of the chest, and upon the flanks. The face is hardly ever affected beyond where the forehead and scalp join. At first the spots are bright red, but afterward become livid and coppery. Some of them are level with the skin, while others rise slightly above it. In the latter case they resemble the wales of urticaria, although, unlike the latter, they do not itch. After lasting a long time, the roseola spots assume a dirty brownish-red appearance, and, when they finally fade, they leave a grayish-brown stain behind them. Sometimes the eruptions extend to the glans penis. In the latter situation, the epidermis over the efflorescence soon separates, leaving a bright-red, moist erosion, which bleeds readily, and which is not to be confounded with the superficial chancre. The roseola is one of the earliest symptoms of constitutional infection. It is never seen in inveterate cases. When treated by mercury, it usually subsides within four weeks. When neglected, it passes over into the papulous, squamous, and pustulous forms of syphilide. The papulous syphilide (lichen syphiliticus) is characterized by its coppery-red, and frequently, in old cases, by its brownish-red color. The papules are sometimes no larger than a millet-seed (miliary papules); sometimes they attain the size of a lentil (lenticular papules); sometimes they are solitary; sometimes they form groups, which latter usually assume the form of circles, or the segment of a circle. Their most common seat is the junction of the forehead with the hairy scalp, although they also appear upon the trunk and extremities. When of long standing, they are usually covered with scales of detached epidermis. If, instead, small pustules form upon the apices of the papules, the eruption is generally called acne syphilitica. Like the maculous syphilide, the papulous exanthema appears early in the disease, although it is sometimes observed in its later stages. In such cases, the points of efflorescence are less numerous, and evince a still more marked tendency to form circles or arcs of circles. The papulous syphilide is

more intractable than the maculous, and several weeks usually elapse before it yields even to an energetic treatment.

The scaly syphilitic eruption (psoriasis syphilitica) often commences as a syphilitic roseola or lichen. The spots are discrete, and rarely become large, although they may be very numerous. The color of a specific psoriasis is generally darker than non-specific, and the scaly layer is usually thin. Not unfrequently cracks form in the infiltrated skin, which lead to ulceration. As we have already remarked, the knees and elbows are hardly ever affected in syphilitic psoriasis. Palmar and plantar psoriasis, which always is of syphilitic origin, begins with the formation of small, round, or oval callous spots of a pale-red or yellowish color. After the thickened cuticle has become detached from these spots, or has been scratched off by the patient, the copper-colored, infiltrated cutis is exposed to view, surrounded by concentric circles of dried desquamating epidermis. This circle enlarges, while sometimes the centre begins to heal, or covers itself afresh with a coat of horny cuticle. Now and then the efflorescences, which at first were separate, afterward coalesce, causing great thickening of the epidermis, which cracks readily, forming extremely painful fissures or rhagades.

The pustulous syphilide is called impetigo, or ecthyma, according as it produces small and pointed or large and flat pustules. They are sometimes solitary, sometimes formed in groups, and appear upon the face and scalp as well as upon the body and extremities. They are surrounded by a coppery areola, and dry up into scabs, which, when situated upon the scalp, are very annoying, owing to their liability to be torn off by the comb. Beneath the scabs of the ecthyma pustules, and more rarely under those of impetigo, there are ulcers, which eat more or less deeply into the cutis. The color of the scars, which always remain after the healing of ecthyma, is at first a coppery-red, and afterward remarkably white. Syphilitic impetigo and ecthyma are more serious affections, and appear at a later stage of the disease than any of the exanthemata hitherto described. In spite of the most careful treatment, many months often elapse ere they begin to heal. The pustulous syphilides are quite intractable, and sometimes outlast all the other symptoms of the disease.

There is an eruption known as varicella syphilitica, and which, indeed, bears a great resemblance to varicella. It is almost always preceded by febrile disturbance. The vesicles, which are numerous, and scattered more or less over the whole body, spring from red maculæ. The liquid contained in them is at first slightly turbid, afterward purulent, and finally dries up into round, blackish crusts, which, when they fall, leave brownish stains behind. This, also, is an obstinate

variety, and often drags on with repeated relapses for weeks and months.

Rupia syphilitica, like ecthyma, originates in a destructive dermatitis. Its mode of development is as follows: Upon a livid, red spot, of the size of a pea or bean, there rises a flabby bleb, containing a dirty, turbid, and sometimes bloody liquid. The contents of the bleb dry up into a scab, which gradually is built higher and higher by the product of the ulceration, which is constantly eating deeper into the skin beneath it, while a ring of new vesicles forms around its border, the drying of whose contents makes the scab wider. If we detach one of these oyster-shell-like crusts, we find beneath it a foul, ulcerated surface, secreting a thin ichor. Sometimes large portions of the body are studded with numerous rupia scabs, which here and there are confluent. In other instances, there are only a few, which then are very large. Like ecthyma, rupia belongs to the graver affections, and only appears at an advanced stage of the disease. They heal very slowly. Not unfrequently, only one side of the ulcer beneath the scab heals, while on the other it continues to spread. In this way, horseshoe-shaped, or kidney-shaped ulcers form. The scars, which always result from rupia, are like those of ecthyma, only larger.

While the syphilitic cutaneous affections hitherto described are the result of irritative and inflammatory processes, syphilitic lupus depends upon the development and degeneration of a neoplasm peculiar to this disease, which arises in the form of nodules ("tubercula syphilitica") not merely in the skin, but in a variety of other organs. These syphilitic tubercles (called "gummata," or "gummy tumors," by *Virchow*, even when they are of a hard consistence, and remain so while they exist, and which are called "nodular syphiloma" by *Wagner*) have nothing in common with tubercle in the common sense of the word. *Virchow* counts them in the class of "granulation tumors," that is, tumors which even at their fullest stage of development contain no mature connective tissue, nor any analogue of it, but consist mainly of elements of a transitory nature, and in which degeneration, death, softening, and ulceration is the regular and necessary consequence of existence. Wherever the syphilitic tubercle appears, it consists of nests of very numerous small cells, with large nuclei, which are lodged in the interstices of the affected tissue, and from which they have sprung through profuse multiplication of its cellular elements. The recent nodules are soft, of a grayish-red color, and infiltrated with a scanty juice. After lasting for some time, they either soften and ulcerate, or else undergo an incomplete cheesy metamorphosis. The most frequent seat of the syphilitic cutaneous tubercle is upon the face, especially upon the forehead (corona veneris), and, next in frequence, upon the

region of the shoulder-blades, and the dorsal surface of the extremities. Some of the nodules are superficial, while others are deeply seated. The more superficial ones are smaller, while the deeper seated ones are larger. At first a small, movable tumor, somewhat sensitive to pressure, appears in the skin, which gradually reddens above it, and rises into a dark-red hemispherical nodule, which rather resembles a boil. Sometimes the covering of the tubercle remains unbroken, and, its contents becoming absorbed, its surface gradually grows paler, sinks in, and a scar forms without there ever having been any ulceration. If the tubercle breaks, a turbid, serous liquid escapes under the cuticle, and, thickening, forms with it a scab. Beneath this scab the ulcer continues to penetrate more and more profoundly. Not unfrequently one common, broad crust covers a cluster of nodules. The ulcer may retain the circular shape of the tubercle, but it more commonly happens that the sore extends at one edge, while it granulates and cicatrizes at the other. Thus, like the rupia, the tubercle produces horse-shoe and kidney-shaped ulcers. The scars, which remain after healing of a syphilitic lupus, long retain a brownish-red stain, and, after the disease has subsided, become remarkably white.

The hair often falls out, in syphilis; but, as a rule, this is merely a defluvium capillorum, unless the scalp be the seat of an eruption. The hair-bulbs do not perish, and, after the disease has abated, the growth of the hair is as luxuriant as ever. The syphilitic exanthemata, on the other hand, usually destroy the hair-follicles, and cause permanent baldness or alopecia.

The nails, also, undergo changes from implication of their matrix in the cutaneous disorder. In the simple infiltration of the matrix of the nail, which often accompanies psoriasis syphilitica, the nails degenerate, becoming misshapen, fissured, and horny. In the more rare ulceration of their matrix, they often become detached.

V. *Syphilitic Disease of the Mucous Membranes.*—Next to the external integument, the mucous membranes are the most common seat of syphilitic disease, although all of them are not affected by constitutional syphilis with equal frequence. The lining of the mouth, fauces, nose, and larynx, and, next to these, the mucous surface of the rectum, are the regions upon which the malady is most prone to localize itself.

The mildest form of syphilitic disease of the mucous membrane, and at the same time the one which appears soonest after the primary infection, is syphilitic catarrh. In special instances, it is not always easy to distinguish between a catarrh depending upon syphilis, and one arising from other causes. A remarkably deep, bluish redness, an abrupt line of demarcation between the affected spot and the sound

parts, and a milky, turbid appearance of its epithelial coating, are suspicious, but not pathognomonic signs. In most cases the diagnosis depends mainly upon the history of the case, and upon the coexistence of other syphilitic symptoms, particularly enlargement of the lymphatic glands, and maculous or papulous exanthemata, and above all upon the refractoriness of the catarrh to simple treatment, and its speedy disappearance when treated by mercury.

The appearance of mucous papules, with their transformation into condylomata, erosions, and ulcers, is one of the earlier symptoms of syphilitic infection. Their mode of development is as follows: A spot of mucous membrane of about the size of a pea rises above the surrounding level, and seems firmer, and somewhat reddened. If it does not heal after reaching this stage, the epithelium covering the flattened nodule becomes opaque, and assumes a milky, or pearly appearance. After separation of the thickened epithelium, a bright-red erosion remains, which bleeds easily. This is followed by a sore, with an uneven, whitish-gray bottom, the product of molecular disintegration, or else plaques muqueuse and mucous condylomata form, from augmented vegetation and development of connective-tissue fibres. We have already treated of the syphilitic mucous papule, and of the ulcers and condylomata of the mouth, fauces, and larynx, to which they give rise. We rarely obtain an opportunity of observing the earlier stages of syphilitic disease of the rectum. The ulcers which arise from mucous papules of the rectum, and the scars which they leave after healing, bear a close resemblance to dysenteric scars. A distinction may be based in some measure upon the seat of the sore, and resulting cicatricial stricture, the syphilitic ulcers being situated in the cloaca of the gut, or else close to the anus, while the dysenteric ones are found in the sigmoid flexure.

The gummata and tubercular syphilomata of the mucous membranes have the same character and mode of development as those of the skin. A nodule of the size of a shot forms in the mucous membrane, and gradually grows until it projects above the surrounding level. If not made to resolve itself by judicious treatment, it softens, bursts its epithelial cover, and turns into a sore. Wide-spread destruction of the nose, palate, fauces, and larynx, may thus occur. Not unfrequently the disease spreads to the submucous tissue, and the cartilage, periosteum, and bones. The ulcers often produce contractions in healing.

VI. *Syphilitic Iritis.*—The syphilitic contagion not unfrequently gives rise to inflammation of the iris and the choroid coat of the eye. This is a very common complication of the syphilitic exanthemata. Modern ophthalmologists deny the existence of any specific mark of distinction between the syphilitic and non-syphilitic inflammations of

these membranes—such as distortion of the pupil inward and upward. Besides the simple syphilitic iritis, there is another form, known as "gummous" iritis. For a description of the very characteristic symptoms and course of this affection, we must refer to the text-books upon ophthalmology.

VII. *Syphilitic Disease of the Periosteum and Bones.*—One of the common symptoms of syphilis consists in a pain along the bones, unaccompanied by any appreciable objective manifestations. At first these pains are vague and ill-defined. Afterward they are fixed at certain points, particularly in the bones lying close beneath the skin, such as the tibiæ and the cranial bones. They are increased by pressure, and usually remit during the day and recur at night. These *osteocopic* pains are probably dependent upon a slight disease of the periosteum, capable of complete and speedy recovery, and which probably consists in moderate hyperæmia with an inflammatory œdema. The severe strain which the naturally-unyielding periosteum suffers, even when but slightly infiltrated by serum, fully accounts for the violence of the pain.

In bad cases of inveterate syphilis, swellings form here and there upon the bones, accompanied by great suffering, which becomes excruciating at night. Such swellings, when of a boggy consistence, are called "gummata;" if their consistence be somewhat hard, they are colled "tophi" (nodes). Like all other syphilitic affections of the periosteum and bones, they are most apt to appear upon the skull, the shins, the sternum, and other bones lying close beneath the skin. Gummata receive their name from the viscid-looking liquid which flows from them when punctured. They consist of cells and nuclei, with very little connective tissue, and a great deal of liquid intercellular substance, their elements corresponding closely to those of the recent succulent syphiloma. These tumors may decrease and disappear entirely, the liquid and cells both being absorbed, the latter first undergoing fatty degeneration. In other instances pus forms in them, and they turn into abscesses, which, when opened, either naturally or artificially, discharge their contents.

Tophi, although their consistence is very hard from the outset, so that they are apt to be mistaken by ignorant persons for excrescences from the bone, are circumscribed neoplastic thickenings and elevations of the periosteum by inflammatory exudation. The periosteum whence they spring has but little tendency to suppuration, and by means of an early and appropriate treatment we can often bring about their resolution. When of longer standing, bone forms in the thickened tissue; the tophi are thus converted into exostoses, and are then no longer capable of reabsorption. Besides the form of exostosis just mentioned,

which proceeds from ossification of the periosteum, there is another, dependent upon inflammation and proliferation of the bone itself. Tophi and exostoses by pressure upon neighboring nerves may cause neuralgia, anæsthesia, or palsy, and when situated upon the inner surface of the skull may occasion serious disorder of the brain.

Syphilitic caries and necrosis are sometimes the consequence of purulent periostitis, where the pus effused between the bone and periosteum has cut off the portion of bone affected from its nutrient blood-vessels. At other times it proceeds from an ulceration, originating in the soft parts, and which has destroyed the periosteum, and laid bare the bone; while, in still another series of instances, the periosteum, at first, is healthy, and the caries and necrosis are the result of a simple or gummy syphilitic inflammation. The question why the denuding of the bone of its periosteum and the ostitis sometimes cause an ulceration of the bone, or caries, while, at others, it induces the death and separation of large pieces of bone, or necrosis, we shall leave undiscussed, as being a question belonging to general surgery. When bone exfoliates from syphilitic necrosis, the loss of substance is seldom filled up by new bone. A depressed spot almost always remains, or, if the bone has been perforated, there will always be a hole in it with smooth edges. Syphilitic caries and necrosis occur more frequently upon the bones of the face and skull than upon those of the trunk and extremities, although the latter, especially the sternum, tibiæ, and clavicles, do not always escape. The most dreadful devastation occurs in the bones of the nose and hard palate. The vomer and the vertical plate of the ethmoid usually go first, so that the two nostrils communicate by an orifice of variable size. Afterward the entire bony septum perishes, and the turbinated bones, the walls of the ethmoid cells and frontal sinus, the nasal and lachrymal bones, are also destroyed. The nose thus loses its support, and sinks in behind its apex, which then points upward. If the disease attack the floor of the nostrils, after perforation of the hard palate, a communication is established between the cavities of the mouth and nose; so that food and drink can enter the latter. More rarely, destruction of the hard palate commences in the mouth, although I have seen a piece of bone as large as a florin exfoliate from the lower surface of the hard palate, without any syphilitic ozæna. When caries and necrosis of the nasal bones arise from ulceration of the mucous membrane which has penetrated and destroyed the periosteum, the patients suffer long from an ill-smelling ichorous and often bloody discharge from the nose, which, at a later period, also contains bits of blackened bone. The septum of the nose is often perforated in such cases without its external covering exhibiting any particular change; but, if the destruction of the nasal bones be

preceded by syphilitic periostitis, the skin soon becomes reddened, and the face grows œdematous upon the affected side. Sometimes the integuments are perforated by the pus and fragments of bone, and thus a fistulous opening is established in the back of the nose. Syphilitic ozæna, often causing a more or less extensive destruction of the bones of the nose, may recover completely. Some time ago, at the autopsy of a syphilitic person, besides an extensive recent anostosis excentrica (see below), I found a complete absence of the bony septum, the turbinated bones of the nose and the inner walls of the antra of the upper jaws. But there was no bare bone to be found, the huge cavity which represented the nose being everywhere lined by a smooth cicatricial membrane.

Besides caries and necrosis, a third destructive disease of the bones occurs in syphilis, which was first accurately described by *Bruns*, although *Virchow* first pointed out how frequently it originated from a syphilitic taint. *Bruns* describes this peculiar form of consumption of bone, to which he gives the name *anostosis excentrica*, as a dissolution and liquefaction of the bone, beginning excentrically, proceeding from the medullary canals and medullary cells, and marked by swelling and redness of the tissues contained in these spaces, but never accompanied by suppuration. The calibre of the canals is first enlarged at the expense of their walls, the progressive absorption of which ultimately causes the canals to blend into irregular cavities. The effect of this process is to render the diseased bone porous, and spongy, like carious bone, and in a macerated specimen it is impossible to tell whether the destruction is the result of caries or of anostosis excentrica. When the disease commences upon the exterior of the skull, its surface first assumes the worm-eaten, rough appearance of superficial caries; but afterward the loss of substance extends more deeply, and here and there may even perforate the cranial wall. The inner surface of the pericranium is said to be reddened, swollen, and granulated over the whole diseased region, and the medullary tissue within the diseased bone is converted into a red vascular mass, intimately connected with the pericranium. Not a trace of pus is anywhere to be found. The adjacent bone is either unaltered, or else a reproduction of bony matter takes place on the edges of the seat of disease, which is thus surrounded as by a wall. When the process attacks the outer surface of the cranial wall, an extensive growth of new bone may also occur on the interior of the vault of the skull. This description of anostosis excentrica, which has been taken almost word for word from the text-book of *Bruns*, agrees so closely with *Virchow's* account of "caries sicca," or the inflammatory atrophy of the bones of syphilitic persons, that it can hardly be doubted that both observers have described one and the

same disease. From microscopic examination of the soft tissue and of the more solid and resisting masses, the points of which contain a yellowish-white and very dry substance, *Virchow* has satisfied himself that the conical or spindle-shaped contents of the cavities, resulting from the loss of bone, consist of the specific neoplastic product of syphilis, to which he gives the name of gummata even when it is not soft, and which *Wagner* calls the syphiloma.

VIII. *Syphilitic Sarcocele.*—Syphilitic contagion sometimes gives rise to an inflammation of the testicle, distinguishable from other forms of orchitis by certain peculiarities, particularly by its seat, and the tediousness of its course. The disease begins in the tunica albuginea and its continuations, and induces a proliferation of young cells and new connective tissue upon the interior of the tunic, and between the seminal tubules. Under the pressure caused by this tumor, which afterward contracts and indurates, the proper substance of the gland disappears. Besides this simple syphilitic orchitis, there is a second form, distinguished by the formation of dry, whitish-yellow, homogeneous nodules in the affected organ, in addition to the proliferation of the albuginea. *Virchow* calls the former disease simple syphilitic orchitis, and the latter, gummy orchitis. *Wagner* believes that there is no essential difference between the nodular syphiloma, or gummy tumor, and the diffuse syphiloma. According to his views, the form first mentioned is the diffuse syphiloma, while the latter constitutes the nodular syphiloma of the testicle. The symptoms of syphilitic sarcocele consist in a slowly-progressing enlargement of one or both testicles, which develops either without any pain at all, or else is accompanied by occasional lancinating twinges. The testicle becomes very hard, loses its regular shape, and may grow to the size of a fist. Sarcocele is often accompanied by a serous effusion in the tunica vaginalis propria. Sometimes the disease, after making some progress in one testicle, attacks the other.

Virchow describes a syphilitic *periorchitis*: This may commence as a hydrocele, but soon occasions thickening of the albuginea and tunica vaginalis propria, causing adhesions, or even complete synechia of the latter.

IX. *Syphilitic Disease of the Connective Tissue, Muscles, and Viscera.*—In cases of inveterate syphilis, tumors presenting the characteristics of the gummy tumor, or syphilitic tubercle, are also found in the subcutaneous and submucous connective tissue, and in the interstices of the muscles (*Virchow*). When properly treated, they sometimes disappear, while at other times they suppurate, producing abscesses and intractable ulcers.

The muscles also, especially those of the upper extremity, the throat and neck, are sometimes the seat of syphilitic disease. *Virchow*

describes a simple and a gummy myositis. The former depends upon proliferation of the interstitial connective tissue, producing an induration of the latter, while the primitive muscular fasciculi suffer atrophy, and perish. In the latter form, tumors, often of considerable size, arise in the muscles, and upon section show reddish-white or yellowish-white, slightly striped deposits, which are usually not absolutely dry. Microscopic examination reveals a finely-cellular, dense granulation of the intramuscular substance, with premature fatty degeneration, in which the cells perish, leaving a granular, fatty, and apparently structureless mass. Similar masses are sometimes found in the substance of the heart. Perhaps some cases of induration of portions of the cardiac muscles depend upon simple syphilitic myocarditis.

Among the syphilitic diseases of internal organs, syphilitic hepatitis has been described in detail. *Wagner* and other authorities have observed syphilitic lesions in other organs, the spleen, the kidney, and even the pancreas, which closely corresponded with those found in the liver, sometimes inducing thickening in the envelope of the viscus, sometimes diffuse induration of its substance, and sometimes producing circumscribed nodules in it. *Dittrich* has already recognized the occurrence of syphilitic disease of the lung, in the form of nodular, firmly-resisting indurations, either of a white color, or else stained black, by deposit of pigment, and which enclosed dry, cheesy accumulations. It would also seem that, besides this gummous pneumonia, an interstitial syphilitic pneumonia also occurs. It may sometimes be very difficult, or even quite impossible, to decide whether a disease of the lung depends upon syphilis or not.

Syphilitic affections of the encephalon consist in part of chronic inflammation of the meninges, which may involve the pia mater, the arachnoid, or the dura mater, in the form of a pachymeningitis externa (endocranitis) or interna; and, among other symptoms, sometimes give rise to palsy of one of the cerebral nerves. Sometimes, syphilitic deposits occur, which formerly were often mistaken for inspissated abscesses, or for caseous tubercles. (See "Diseases of the Brain.")

It follows, from this brief enumeration of the various syphilitic diseases of the tissues and organs, that the points on which the disease localizes itself are far more numerous than was formerly supposed; and that *Wagner's* statement, that syphilis attacks all the tissues which contain vessels, is fully warranted.

The General Course of Syphilis.—It is a remarkable fact that, while the syphilitic taint at times gives rise to palpable symptoms, at others no sign of the persistence of the infection can be detected, excepting the enlargement of the lymphatic glands After the healing of the primary ulcer, a period of several weeks usually elapses before

condylomata, exanthemata, or other consequences of the infection, appear; and, after the latter have subsided, another interval of exemption usually ensues, ere a second series of disorders disturbs the apparent good health of the patient. For these alternations, which usually recur again and again during the course of the disease, no satisfactory explanation has as yet been furnished. The time which intervenes between the healing of the primary ulcer and the appearance of the secondary symptoms, as well as that between the various outbreaks of the secondary manifestations themselves, varies greatly in different cases. The causes of this difference, and the influences which shorten or prolong the period of latency, are likewise in a great measure unknown. It seems, however, that in vigorous constitutions, and in persons who live luxuriously, the secondary symptoms set in sooner, and recur at shorter intervals, and that the length of the period of latency is somewhat dependent upon the treatment adopted.

Bärensprung, relying upon a large experience, affirms that, when mercury is not employed, a period of latency of several months, or even years, never occurs, and that the secondary symptoms and their relapses appear within six weeks at latest, after the healing of the indurated ulcer, or after the subsiding of the outbreak last treated. He does not hesitate to declare a patient treated without mercury to be safe and permanently cured, who continues free from constitutional symptoms three months after treatment has been discontinued. If this assertion could be substantiated, the well-authenticated cases of latency of syphilis, during a period of ten or twenty years, would be attributable entirely to mercurial treatment. Such protracted periods of latency, however, if they really do occur, are at all events extremely rare, be the treatment non-mercurial or mercurial. It is equally rare, also, for secondary symptoms to arise prior to the healing of a recent primary sore. As a rule, they do not make their appearance until from eight weeks to three months after infection, and the relapses usually follow one another at similar intervals.

A certain degree of regularity can be recognized in the manner in which the various manifestations of the disease succeed one another After the healing of an indurated ulcer, there is a great probability that in a few weeks or months it will be followed by condylomata, a maculous exanthema, or by a syphilitic angina; but, at this stage, the patient is quite secure against rupia, syphilitic lupus, or disease of the bones. Conversely, an individual, who for years has suffered repeated relapses of syphilis, is in danger of destructive disease of the skin and subcutaneous areolar tissue, of caries and necrosis of the bones, but not of broad condylomata, or of syphilitic roseola. The disorders and complications of disorders which stand first in order in the series of

syphilitic affections are called *secondary* affections; others, which are likewise frequently combined one with another, but which appear later, are called *tertiary* affections. The former are usually considered to include indolent buboes, the condylomata, the exanthemata with the exception of rupia and lupus, the superficial ulcers of the mucous membrane, and iritis; while lupus, rupia, disease of the bones, and the gummy tumors of the submucous and subcutaneous areolar tissue, and the disorders of the muscles and viscera, belong to the tertiary class. Upon comparing the two classes, it will be found that the secondary diseases are less malignant, that is, are less destructive than the tertiary; and that they usually are limited to the superficial tissues, to the skin and mucous membrane; while the tertiary affections attack the deeper-seated, "more noble" organs. This mode of classification, however, makes it impossible to draw a sharp boundary between the two forms, and it is idle and unprofitable to dispute as to whether the intermediate varieties rank as secondary or tertiary, such as sarcocele, which, though not one of the earlier consequences of infection, still usually precedes lupus and disease of the bone, and which, in respect to its malignity and to its locality, occupies an intermediate position. In rare and exceptional instances, disease of the bones forms one of the early symptoms of general syphilis. The opponents of the treatment by mercury attribute this early appearance of the so-called tertiary accidents to the mercury, and even go so far as to assert that the employment of mercurials, by aggravating the pernicious influence of syphilis upon the organism, actually is the cause of the tertiary manifestations; and that, under non-mercurial treatment, syphilis does not affect the bones. Although both statements, in their fullest acceptation, are decidedly untrue, yet neither of them is quite without foundation; for, if the disease be not subdued, while the constitution has been ruined by immoderate and reckless dosing with mercury, pernicious forms of syphilis, such as lupus and disease of the bones, are more liable to arise than when it still lurks in the system of a robust individual. It would almost seem, indeed, that the constant increase in malignancy which shows itself in each succeeding attack, in cases of syphilis where mercury has not been employed, is the result of deterioration of the constitution induced by the previous attacks. At all events, the contrary effect is seen in robust constitutions, in whom, when the disease is not completely extinguished at first, each relapse is less severe than the preceding one.

While, with the exception of a slight fever, which usually precedes and accompanies the secondary symptoms, the disease is borne for a considerable length of time without constitutional disturbance, yet, when the attacks recur constantly, when the patient's rest is continually dis-

turbed by nocturnal pain, when protracted suppuration consumes his strength, and, above all, if an active mercurial treatment be superadded, a general marasmus, the so-called syphilitic cachexia, sets in. This term is not exactly appropriate, since it does not proceed directly from syphilitic infection, but indirectly from the nutritive disturbance to which it leads, or from the method of treatment adopted. Accordingly, the syphilitic cachexia has no peculiar features by which it may be distinguished from other non-specific forms of cachexia. Even amyloid degeneration of the liver, spleen, kidneys, and other organs, which often accompanies the syphilitic cachexia, is not peculiar to this disease, but also develops in the course of other non-syphilitic diseases of bone as well as in malarious affections, scrofula, and rickets.

Treatment.—The treatment of the primary syphilitic induration and primary syphilitic ulcer must be directed mainly against the constitutional disease, the first manifestation of which we have found it to be. Abortive treatment is out of the question, save when we have to treat an excoriation produced during a suspicious coitus; but in such a case, after destroying the affected part, we cannot tell whether the absence of induration and of subsequent symptoms is the result of the treatment or not, as we have no criterion for distinguishing an infected excoriation from a non-infected one. The extirpation of an induration, or its destruction by means of caustics, is altogether useless. As a general rule, a new induration forms upon the edges of the wound thus produced. Besides the use of constitutional remedies, the primary syphilitic sore should be treated according to the principles already laid down for treatment of the chancre. First of all, the utmost cleanliness must be maintained. Washes, mild or strong, according to circumstances, should be used, together with gentle applications of lunar caustic, or sprinklings with red precipitate, and the like. Internally, unless contraindicated by special circumstances, I treat the primary ulcer and primary sore with mercurials. I am well aware that the induration is capable of disappearing without their use, and that this treatment affords no guaranty against the appearance of consecutive symptoms; but the indisputable fact, that the secondary symptoms are fewer and later in appearing when treated by mercury than when treated without it, induces me to prefer the former treatment to the latter. Besides, a cautious employment of this drug is far less liable to be followed by mischievous results than is asserted by its opponents. In the first year of my practice I did not employ mercury in syphilis, but in my second I commenced to use it upon almost every indurated sore, and in all cases of secondary disease. The number of patients which I have treated in this manner cannot of course be compared with that treated in the course of a year or two in the syphilitic ward of one

of the great hospitals, but it is large enough to prove that the mischief produced by mercurial treatment has been greatly overestimated. As I have kept most of the patients, whom I have treated for syphilis, under observation for some time afterward, and since, after the marriage of many of them, I have become their family physician, it would have been more difficult for the pernicious effects of a course of mercury to escape my notice, than that of many a chief of a large syphilitic ward, who loses sight of his patients as soon as they are discharged. A series of observations, many of them very close ones, and now continued for over eleven years, of a by no means inconsiderable number of persons who have undergone a careful mercurial treatment, has converted me from an opponent into a decided advocate of mercury.

It lies out of the plan of this work to recount all the celebrated modes of treatment by mercury, or to detail the minute directions enjoined in the different methods, the number of pills to be taken daily, the manner of raising and reducing the dose, etc. I admit that I regard all sharply-defined routines not only as useless, but as absolutely dangerous, as it induces inexperienced or careless physicians to treat all varieties of constitution by the same formula. Experience has taught that the efficacy of mercury, as a remedy against syphilis, does not depend upon the form in which it is administered, whether as a suboxide, an oxide, a basic salt, a chloride, or an iodide. It is equally immaterial, as regards its efficiency as a remedy, how the mineral enters the blood, whether it be by the intestinal mucous membrane or through the skin. Hence our choice of a preparation of the drug should fall upon one which, while an effective remedy, does the least possible harm to the constitution. Since, however, the pernicious influence which this mineral exerts upon animal life is susceptible only of a partial explanation, we can merely diminish or avert the more subordinate mischief to the intestinal mucous membranes, to which the use of mercurials gives rise, by choosing preparations which are the least irritating to them, or by not introducing the drug through the intestines at all, but through the skin. If we deemed the action of calomel and iodide of mercury upon the intestine, when cautiously administered, to be very great or very pernicious, we should regard the reintroduction of the "inunction-cure" as a most important step in the therapeutics of syphilis; but, believing as we do, that these disadvantages are trifling, and nearly always temporary, we cannot concur in the ecstasies of those who see the beginning of a new era in the reëstablishment of the treatment of syphilis by mercurial inunction. I do not mean by this that I look upon mercurial inunction as an improper method of treating syphilis; indeed, I very often make use of it myself, especially in clinical practice, for it does all that mercurial treatment is

capable of doing; and I know of no objection to it, excepting that salivation sometimes occurs more suddenly and with greater severity in this than in other cures. As a rule, I follow the directions laid down by *Sigmund* for this mode of treatment (which, compared with the notorious and dangerous "grand inunction-cure" of *Louvrier*, scarcely deserves the name of inunction), but I do not pedantically hold myself strictly to *Sigmund's* precepts. The patient having taken several daily warm baths, I cause a half drachm of blue ointment to be rubbed into the skin, and, where there seems to be danger from delay, I rub in a whole drachm. Upon the first day I apply it to the legs, the second to the thighs, the third to the arms, the fourth to the back, the fifth to the legs again, and so on. The precise order in which the rubbings succeed one another is of course unimportant. At points within the patient's reach he can rub the ointment in for himself; at others, an attendant must do it. Each inunction should continue from ten minutes to a quarter of an hour, and, before rubbing again, the surface previously rubbed is to be washed with soap and water. The temperature of the room should not be above sixty-five or seventy degrees Fahrenheit, and must be ventilated daily. There is no objection to the patient's changing his linen. It is useless to combine the hunger-cure with the inunction-cure; and it may even do harm. As soon as the least trace of salivation shows itself, the treatment must be suspended; and any of the ointment still adherent to the skin should be carefully removed by a bath, or by a thorough washing. Should the healing of the ulcer or resolution of the induration come to a stand-still after cessation of the salivation, or, in cases of secondary disease, should the improvement not continue after the salivation abates, I recommence the mercurial frictions, one or two of which then almost always perfect the cure. I observe the rule of always suspending the treatment upon the occurrence of salivation, not only when the mercury is employed in the form of ointment, but in all other forms of mercurial treatment, and simply because I regard salivation as a sign that a sufficient quantity of the mineral has been absorbed. It matters not whether the mercury be applied upon the skin, or to the intestines; in either case a part only of what is administered is absorbed, and the rest either remains upon the skin or else is expelled in the stools. Hence, even if we could tell how large an amount of it must be introduced into the system in order to effect a cure of a syphilitic affection, we should still be ignorant of the number of inunctions of ointment, or of internal doses of calomel, corrosive sublimate, or iodide of mercury, requisite to produce the desired result. Salivation, of course, affords no criterion of the amount of mercury absorbed, but it certainly indicates that enough of the drug has been taken up to pro-

duce a decided impression upon the system. That the one effect, namely, the salivation, is almost always accompanied by another, the improvement of the syphilitic lesion, follows even from the erroneous belief entertained by many old physicians, that salivation is a beneficial crisis. Notwithstanding that I regard salivation as a valuable guide in the treatment of syphilis by mercury, just as I regard dilatation of the pupil as valuable in treatment of epilepsy by atropin, showing that the dose administered is sufficient, and although I do not regard the appearance of slight traces of salivation as an objection to such method of cure, yet I consider it extremely unsafe when we have not the opportunity to suspend the treatment before the salivation has made any considerable progress. Salivation has no curative effect whatever, and I always seek to allay it by administration of chlorate of potash, either in solution (℥ j to water ℥ ij), a tablespoonful of it to be taken every two hours, or else in the form of pastilles. If, when in doubt whether to continue the cure or to discontinue it, we decide to give one or two more doses of calomel, corrosive sublimate, or iodide of mercury, we run far less risk, by so doing, of producing a severe sore mouth, than if we rub in another half drachm of mercurial ointment.

The great objection to the inunction treatment, that in it we cannot say how much of the mercury enters the body, does not exist in using *subcutaneous injections of corrosive sublimate.* In this treatment, which of late has run a strong opposition to the inunction treatment, and has to some extent supplanted it, we may even more certainly control the dose than we can in internal administration, and we save the gastric and intestinal mucous membrane just as completely as we do by the inunction treatment.

I acknowledge that I began the employment of subcutaneous injections of corrosive sublimate with a certain distrust, and that I did not resort to it till it had been urgently recommended to me by various persons. I feared that the pain and inflammations, with partial necrosis of the skin that it must cause, would be out of proportion to any possible benefits arising from its use; indeed, it seemed doubtful to me if this inflammation and necrosis of the skin would not interfere with or entirely prevent the absorption of the remedy. Experience soon taught me better. If sufficiently dilute solutions are employed, the pains are perfectly bearable and of short duration; by the same precaution, severe inflammations and necrosis of the skin may be avoided. The absorption of corrosive sublimate appears to follow subcutaneous injection just as quickly as that of morphine does; in short, the advantages seem to outweigh the objections so much, that any practitioner, who has treated his syphilitic patients for a short time with subcutaneous injections of corrosive

sublimate, will acknowledge that we are much indebted to *Lewin* for its introduction. In my clinic, we at first used *Leiter's* syringe, which holds twelve grains of fluid, and with this injected twelve drops of a solution of corrosive sublimate (gr. j to ʒ j). Later, with the best results, we injected a solution (gr. j to ʒ ss) with one of *Paikrt's* syringes, holding half a drachm (that is, the eighth of a grain of corrosive sublimate in a solution one fourth the strength of the former) once or twice daily. We obtained this syringe also from *Leiter* in Vienna, but, as the canula was rather thick, and consequently the puncture pained and bled, we had a canula made like that on the ordinary hypodermic syringe.

Nevertheless, the conviction, that the internal administration of mercurials, cautiously conducted, does not produce any lasting injury to the mucous membranes, has caused me, in spite of the general commendation of injections and the inunction-cure, to adhere to the internal exhibition of mercury in private practice. The article I generally employ in cases of primary induration and primary sore, is calomel. Of all mercurials, this one is the least open to the charge of causing serious or permanent detriment to the mucous membranes when given in moderate doses. If it really were injurious, considering how universally it is employed, especially in diseases of children, the number of persons suffering from its effects would be very large. I will only call to mind that the *Plummer's* powders, used in ophthalmia and other scrofulous complaints, the minute doses of calomel used in infantile diarrhœas, the larger ones, pushed almost to salivation in croup, pleurisy, and other inflammations, still are among the most common of prescriptions, and that ten or twenty years ago they were even more generally administered than now. An exceedingly rich experience has taught us, that the innumerable multitude of persons who have used calomel more or less, for other purposes than antisyphilitic treatment, have not suffered any permanent injury to their digestion or their general health, but are now as healthy as they were before. Calomel, however, has no great reputation as an antisyphilitic (at least, in Germany), and the corrosive sublimate is preferred to it by most practitioners, as a means of treating primary and secondary symptoms The reasons for this are twofold: firstly, it often causes diarrhœa; secondly, it soon produces salivation. I do not regard either of these objections as valid. I have never found that the diarrhœa which nearly always sets in at the beginning of a course of calomel (but which usually subsides in a few days) interferes materially with the cure, and, as salivation soon occurs in spite of it, we may confidently infer that the medicine is not all evacuated, but that a sufficient amount of it is absorbed and taken into the blood. With regard to the second objec-

tion, for reasons given above, I rather look upon the early appearance of salivation as an advantage, insomuch that, in cases where, owing to idiosyncrasy of the patient, salivation occurs later than I had expected, or where it fails to appear at all, I have been anxious lest I might administer too large a quantity of mercury, not having this valuable guide to inform me when to discontinue the remedy. To adult patients I usually give a grain of calomel twice daily, or half a grain three times a day. When given in the form of powder, I have it wrapped in a wafer, in order to screen the mucous membrane of the mouth from contact with the drug. Generally, however, I have it made up with liquorice into pills, each of which contains half a grain. Next to calomel, I have most frequently made use of the protiodide of mercury. I am not aware that this article possesses any advantage over calomel, and I have generally noticed that its exhibition occasions severe pain in the bowels, which scarcely ever occurs in the diarrhœa induced by calomel. On account of these annoying symptoms, *Ricord* (to whose authority the protiodide of mercury mainly owes its reputation and extensive adoption) combines it with narcotics; but, even with Ricord's pills (hydrarg. protiodid., lactucar. gall., āā 3 iss, ext. opii aquæos. gr. ix, ext. guaiac. aquæos. 3 j, f. pil. no. xxxvj), I have often been obliged to give opium, on account of severe pain in the bowels, so that I have now been induced to abandon the use of the protiodide. I shall now merely add a few words about corrosive sublimate, for, although I am by no means one of its admirers, and consider it as far inferior to other preparations of mercury, yet it probably is more generally employed in syphilis than any other. The corrosive properties of the drug forbid its administration in large doses, and I believe a gradual increase of the dose to be improper. Can one expect the stomach to accustom itself to the action of the medicine so as to tolerate large quantities without becoming corroded? and why should we increase the dose when it is of importance to be able to suspend the treatment at precisely the right moment, in order to avoid introducing too much mercury into the system? Corrosive sublimate is valued principally because, in the first place, it is slow to produce salivation, and because, under its use, less mercury is taken into the system than under other mercurial treatment. I cannot regard it as an advantage that the chemical properties of corrosive sublimate should compel us to administer it in such doses that the object aimed at, the introduction of a certain amount of mercury into the system, is only very slowly attained; and I cannot admit that much more mercury is taken up in a treatment by calomel, if discontinued when salivation commences, than in a treatment by corrosive sublimate when pushed to the same point. It cannot be denied, however, that many people are cured of their syphilis by cor-

rosive sublimate, and especially by the very injudicious method of *Dzondi*. If we employ this article, it must not be given on an empty stomach, and is best administered in the form of pills; but they should not be made up with bread-crumbs and sugar, according to *Dzondi's* formula, but with powdered extract of liquorice; and the dose should not be raised, as is done in *Dzondi's* treatment, from the fifth of a grain to a grain and a half, but from half to three-quarters of a grain should be given daily, in divided doses. In order to protect the gastric mucous membrane from the corrosive action of the sublimate, it has been proposed to give the albuminate of mercury, and thus at once to furnish the combination which, when the pure bichloride is used, eventually forms in the stomach, at the expense of the gastric mucous membrane, and experience shows that double doses of this preparation can be tolerated without detriment. If it were not that I am already perfectly satisfied with the medicines already spoken of, I should have recourse to the albuminate. *Bärensprung* proposes the following formula: ℞ Hydrarg. bichlor. corrosiv. gr. ij, ovum unum, aqua destillat. ℥ vj, ammon. muriat. ʒ j, misce exactissime. Filtra. D. S., a tablespoonful every two hours. The dietetic rules to be observed during internal mercurial treatment must be regulated according to the condition of the patient. As a general rule, it is advisable to restrict his diet, without going so far as to let him suffer from hunger; but now and then it may become desirable to feed the patient upon the most nutritious food, under conditions already described in treating of the chancre. The common practice of administering large quantities of "decoction of woods" is superfluous. Finally, while we subject the patient to an active treatment, in order not to expose him to other prejudicial influences, we must regulate his habits and carefully watch over his general health. For this reason it is urgently advised that syphilitic patients, who are undergoing a course of mercury, should keep their room, particularly in winter, and that they should be visited daily.

We have said above that the primary ulcer and primary sore would heal without the use of mercury; but that under such treatment the recovery was slower, and the secondary attacks came on more frequently and earlier. This is equally true of the privation-cure (Entziehungs-cur), and the hunger-cure, the methodical use of cathartic salts, and of *Zittmann's* decoction, and other compound decoctions, the object of which is to throw the skin, kidneys, and intestine, into a state of increased activity, the supply of nourishment, meantime, being reduced to a minimum. If this kind of treatment be very energetically conducted, and if the state of nutrition be depressed by a severe course of simple or double *Zittmann's* decoction, we may, no doubt, succeed

in accelerating the healing of the primary affection. But, frequently, just as the patient is beginning to recover from his eleven or twenty-two days of fasting, purging, and sweating, the first crop of secondary symptoms begins to develop.

The preparations of iodine are altogether useless against the primary affections; although certain physicians, who only practise in the country, or in the smaller towns, and who, rarely having occasion to treat syphilis, have an exaggerated dread of the effects of mercury, place great confidence in iodine. During my practice in Magdeburg, I remember many cases, where travellers visiting the small towns, and consulting the physician of the place for indurated chancres, afterward came under my hands, suffering from the worst iodine eruptions that I have ever seen. From the recipes which they brought with them, it was often quite evident that the iodic exanthema had been mistaken for a syphilide, and the worse it grew, the larger were the doses of iodine prescribed.

The secondary and tertiary symptoms of syphilis must always be treated with the utmost circumspection and care, since there is no disease in which therapeutic errors can do such serious harm as in constitutional syphilis. Mercurial treatment is nearly always of signal benefit in the secondary and tertiary forms of the disease, and frequently not only relieves and allays the symptoms as they arise, but sometimes brings about a complete and permanent cure. If, however, it be administered again and again in unsuitable cases, instead of mitigating the malady, it renders it still more pernicious, causing destruction of the bones, degeneration of internal organs, and even endangering life itself. The fact that horrible forms of syphilis are more rare than they used to be, probably is partially because the "grand inunction-cure" and other methods of over-treatment are being banished more and more from the therapeusis of the malady, and that our employment of mercury is now more cautious and restricted. The indications for mercurial treatment of constitutional syphilis have generally been summed up as follows: The secondary affections alone call for mercury, while the tertiary accidents demand the exhibition of iodine. Although this formula is somewhat inexact, yet, upon the whole, it is perfectly true. Mercurials are indicated in nearly all cases where the affection belongs to the secondary group, while, in cases of a manifestly tertiary character, they are generally contraindicated. To this rule another is very properly added, that even in secondary affections mercury is contraindicated, when it has already been used repeatedly without success. The principles upon which the mercurial treatment of syphilis is based may be deduced from what I have said above, regarding the course of the disease, and from the same remarks it will

also become apparent why I have spoken of the customary formulary of indications as inexact, although right on the whole. When an individual, sooner or later after his recovery from an indurated chancre, begins to suffer from condylomata or from eruptions, I do not hesitate to prescribe mercury, either by inunction with blue ointment, or in the form of calomel, to be taken internally, without regard to whether the indurated ulcer has already been treated by mercury or not, for at this period it very seldom happens that, owing to the state of the patient's general health, or to the form of the syphilitic outbreak (then usually a lupus or an affection of the bones), or from both the causes combined, the constitution of the patient has suffered much either from the effects of mercury or from the disease itself. It is only in these very exceptional instances that I consider a mercurial treatment contraindicated at the period when signs of constitutional infection first appear, for we may be almost certain that, if the syphilis cannot be successfully eradicated by means of mercury, its next attack will be of a most pernicious character. On the other hand, where the constitution is still unimpaired, the good effect of mercury in allaying secondary symptoms is most striking. The condylomata, exanthemata, and sore throat, disappear much more rapidly than an indurated ulcer does, when treated by mercury, and it often happens that not only are the immediate symptoms allayed, but a permanent cure is effected. There are other cases, however, in which the disease only becomes latent and not extinct; and where it reappears some time after the subsidence of the first set of symptoms, or relapses, to use the common expression. The management of these relapses varies greatly, according to their character. When the disorder is slight, as when plaques muqueuses appear upon the tongue, or when a few scattered pustules form upon the scalp, the attack is manifestly of a less serious character than the previous manifestations have been, and it is not advisable to renew the mercurials. Syphilis never subsides suddenly, but usually dies out slowly and gradually, and we may always hope for this favorable termination when the new symptoms are of a less serious nature than their predecessors. Although I do not advise a repetition of the mercurials in such cases, yet the patient should be most carefully watched, kept upon a reduced diet, and preserved as far as possible from all other noxious influences, until the last traces of the disease have disappeared. The "privation-cure" ("Entziehungs-cur"), and even the *Zittmann's* decoction, are suitable remedies for such cases. At the height of the disease, the action of these methods of treatment is merely palliative, but when it is beginning to decline, they really seem to accelerate its complete disappearance. On the other hand, when the relapse shows that the disease has not assumed a milder character, if the new mani-

festations are as severe as the former ones, or even more severe, but still retain the nature of the secondary disorders, and if, as is generally the case, the patient's constitution still seems to be unimpaired, I recommence the mercurial treatment, and, indeed, under certain circumstances (for instance, in case of rapidly-spreading ulceration of the fauces, liable to become complicated with syphilitic laryngitis), I even make the treatment more energetic than before. I order daily inunctions of a drachm of blue ointment, or else give daily, or every other day, two doses of calomel, of ten grains each, after the method of *Weinhold*. It has often been objected, regarding *Weinhold's* treatment, that its only action is a laxative one; that it does not salivate, and that it exerts no important influence upon syphilitic affections. These *a priori* views stand in direct opposition to the results of my own experience in many cases where I have given half-scruple doses of calomel.

If the relapse be unmistakably of a tertiary character, or if it be merely of an intermediate nature between the secondary and tertiary forms; or, if the constitution of the patient have begun to suffer, whether from the disease or the treatment, then mercury is not only contraindicated, but even dangerous. Never use a grain of it under such circumstances, and I am certain that by such precautions the evil effects of mercurial treatment may be averted.

I may be brief in stating the indications for the use of iodine in the treatment of syphilis. In all cases where the spontaneous and speedy extinction of the malady is not to be calculated on, and in which the employment of mercurials is contraindicated, the exhibition of iodine is urgently indicated, and then affords the utmost benefit. If we limit the administration of the medicine to the above class of cases, and are as methodical in its employment as we have been with mercury, we shall find that its value in the treatment of syphilis is by no means inferior to it. The preparation of iodine most generally employed is the iodide of potassium, from ten to thirty grains of which are usually given daily, in the form of a watery solution. Some physicians give larger doses, or strengthen the solution, by adding one or two grains of iodine to it, but apparently do not thereby augment its effect. According to my experience, *Bärensprung* to the contrary notwithstanding, I have found the iodide of iron to be a very efficient preparation, especially where there is advanced anæmia. I usually employ it in the form of syrup of the iodide of iron (℞ syr. ferri iodid. ʒ ij, syr. simpl. ʒ ij. ♏. S. ʒ j every two hours), and by its means have often produced iodic eruption and catarrh. The appearance of this symptom, like that of mercurial salivation, requires a suspension of the remedy. If the suspension be followed by an arrest of improvement in the symptoms, the

iodine must be resumed after the catarrh or eruption has subsided. The diet should not be reduced; not on account of the remedy, however, but because of the condition of the patient's general health. On the contrary, his food should be nourishing, and he should be allowed a little wine or beer. For persons of broken-down constitution I not unfrequently prescribe iron, quinine, and cod-liver oil, besides the iodine.

The local treatment of the secondary and tertiary symptoms should be restricted to the extirpation of old condylomata (which, though they always dry up under mercurial treatment, do not always disappear entirely), to the application of atropine in iritis, to local blood-letting, and to occasional surgical procedures, which may be demanded on account of destructive inflammation of the skin, caries, or necrosis. The majority of the secondary and tertiary affections do not require local treatment.

Syphilization, which, during the last ten years has been much lauded as a certain means of eradicating syphilis, and of protecting the system against further infection, has again fallen into disrepute, or at all events is now merely practised by its introducers, and by the enthusiastic disciples of their new doctrines. The process of syphilization consists in the production of chancre pustules upon the skin of a person afflicted with constitutional syphilis, which process is continually to be repeated, until the inoculation ceases to act. Apart from the fact that some persons do not become proof against the chancre-virus, after suffering several hundred and even as many as two thousand inoculations, it has not by any means always followed that, the immunity once attained, the syphilitic affection has healed; and, where recovery has taken place, it remains more than doubtful whether it has been in consequence of the syphilization.

CHAPTER II.

CONGENITAL AND HEREDITARY SYPHILIS.

Etiology.—We shall pay no attention to the forms of infantile syphilis, arising from the infection of a child during birth by syphilitic ulcers upon the genitals of its mother, or upon the nipples, lips, or other parts of the body, of its nurse, for these forms of syphilis do not differ, either in point of origin, or of course, from the syphilis of adults.

By the term congenital hereditary syphilis, of which alone we now propose to treat, we mean a form of that malady occurring in newly-born children, and originating in the embryo, from constitutional disease existing in the father at the time of begetting the child, or in the

mother during the period of pregnancy. The manner in which the contagion is transmitted to the embryo from the father or mother is altogether unknown, and we shall, therefore, confine ourselves to a statement of a few known and authentic facts, without attempting any explanation of them.

If a woman who has secondary syphilis becomes pregnant, the fœtus nearly always dies prematurely, and is expelled by an abortion or miscarriage. In such cases, the fœtus is generally so much putrefied that it cannot be determined whether it bears traces of syphilis upon it or not. In like manner, a woman who is healthy at the time of conception, but who subsequently contracts syphilis, usually aborts or miscarries, giving birth to a decomposing fœtus. In other cases, the child is carried to term, but dies either at the time of its birth or shortly before. It then either bears distinct evidence of syphilis upon its wasted body, or else there is no anomaly beyond its extreme emaciation. In rare instances the child is born alive, and lives for a longer or shorter period of time. In such cases, the syphilis may appear immediately after birth, or else may remain latent, and develop weeks or months afterward. Inasmuch, then, as constitutional syphilis of the mother exerts so pernicious an influence upon her offspring, that it usually perishes either long before or else during birth, it will be readily understood that the majority of cases of congenital syphilis, which become the object of clinical investigation and medical treatment, are the progeny of syphilitic fathers. It is an extraordinary but well-established fact that syphilis may be thus transmitted from father to child, without infecting the mother who bears the infected offspring in her womb. Hereditary syphilis, derived from a syphilitic father, sometimes manifests itself immediately after birth, while at other times the characteristic symptoms do not appear until later.

Symptoms and Course.—The symptoms of congenital syphilis consist principally of affections of the skin and mucous membranes; and it is only in rare instances, when the disease drags on incurable, but without killing the child, that it causes disease of the bones. When children are born with evidence of the disease upon them, or when it makes its appearance a few days after birth, it usually proves to be of a more malignant nature than when it remains latent for some weeks.

The former class, in which the malady assumes the character of a bullous or pustulous syphilide, occasionally accompanied by a coryza (see below), was for a long time misunderstood, and used to bear the name of pemphigus neonatorum. When the child does not come into the world with the disease already upon him, it usually commences upon the palms of the hands and soles of the feet, and afterward

spreads to the arms, legs, and body, and sometimes to the face. Roundish spots, as large as a pea or bean, and of a dirty-red color, are first observable. In a short time the cuticle which covers them is elevated into a bleb by the effusion of a turbid liquid. These blebs burst, leaving excoriated, moist spots upon the skin, which show no tendency to heal, and new blebs form upon the toes and fingers. Not unfrequently some of the nails fall off. The later crops of blebs behave like their predecessors, and, as, day after day, new ones arise, the unhappy child becomes excoriated at innumerable points. I have even seen the blebs form in the mouth and nose. The malady may last from a week to a fortnight; more rarely three or four weeks, and always terminates fatally. Some of the cases occurring in my private practice, in which the mother, with touching patience, daily cleansed and bound up the fingers and toes of her babe, never suspecting that its dreadful disease was the consequence of the excesses of its father, have made an indelible impression upon my mind.

When syphilis remains latent for a few weeks, it presents another aspect. (The bullous syphilide never commences later than the first week of infancy.) The child is often born in apparently good condition, and does not seem to differ in any respect from a healthy child. About a fortnight after birth, and in some instances as late as a month, or even two months after birth, it grows restless and thin, and its surface assumes a remarkable dirty color. The skin also becomes extremely dry, rough, and shrivelled; and, in particular, the hands and feet look as if covered by a delicate husk, like that of an onion. It soon becomes difficult for the child to nurse, the nasal mucous membrane being swollen, and the nostrils always filled by a thin secretion. This swelling of the mucous membrane, and the profusion of the discharge, give rise to a peculiar snorting and snuffling, which is almost pathognomonic of congenital syphilis; so that, in the absence of other signs, we can hardly err in diagnosticating the disease from this symptom, together with the peculiar condition of the skin. In most cases, the coryza is accompanied by an eruption, which, beginning in the region of the anus, spreads to the genitals, thighs, and loins, as well as to other parts of the body, particularly to the face. This eruption is generally of a character intermediate between that of the maculous, papulous, and squamous forms. The separate maculæ are rounded, of the size of a pea or bean. Their color is a coppery or yellowish red, which becomes actually yellow when pressed upon by the finger. They may be either distinct or confluent. At first they are level with the skin, afterward they project somewhat above it; then their surface is not rounded, but flattened, as if the top had been shaved off. As the affection advances, the maculæ are often covered with scales of epider-

nis, or with a continuous coating of thin desquamating layers of cuticle, and, where the surface is habitually soiled by urine or the excrements, it becomes excoriated. Another constant symptom of congenital syphilis consists in the formation of rhagades at points where the skin changes into mucous membrane, especially upon the mouth and anus. It can often distinctly be perceived that the lips of the child are incurved and bleed readily upon the slightest movement, and that it dreads to use them either in suckling, laughing, or crying. The act of defecation cannot be accomplished without great suffering, so that the child cries and moans whenever the bowels are moved. The rhagades are often accompanied by condylomata, and in neglected cases large ulcers, of a peculiar irregular, angular figure, form between the nates, and sometimes in the flexures of the thighs, and in other regions where intertrigo is apt to arise in healthy children. The discharge from these sores is scanty, and readily dries up into a scab, which is of a blackish color, owing to its containing an admixture of blood. We have already stated that it is rare for congenital syphilis to attack the bones. Cases are reported now and then, however, in which the usually slight ulceration of the nasal mucous membranes causes destruction of the bones of the nose, and depression of that feature in the first year of childhood. In other equally rare instances, congenital syphilis of early infancy either is overlooked or becomes latent in consequence of treatment, and breaks out again at the period of puberty in the pernicious form of syphilitic lupus, or disease of the bones.

In the autopsies of children who have died of congenital syphilis, or who have been born dead by syphilitic mothers, the characteristic lesions of the disease are sometimes found in the internal organs, especially the liver and lungs, more rarely in the brain. In the former it consists merely of a diffuse, uniform induration, the sequel of a simple non-gummous hepatitis. In the lungs, nodules, with cheesy centres of the size of a pea or walnut, are formed, as well as a form of diffuse condensation, first described by *Virchow*, who calls it "white hepatization," and which consists of a filling up of the air-vesicles with epithelial cells in a state of partial fatty degeneration. In the brain, *Schott*, in one case, has found gelatinous tumors of the size of a hazel-nut situated beneath the lower surface of the two anterior lobes of the cerebrum. Finally, the thymus gland is sometimes considerably enlarged, and contains abscesses.

Treatment.—Hitherto, the treatment of the bullous syphilide has always been in vain. On the other hand, in the second form of congenital syphilis just described, comparatively good results may be obtained by means of appropriate medication. The most common and best method of cure consists in the exhibition of small doses of calomel (gr. $\frac{1}{8}$)

morning and evening, and the inunction of five or ten grains of mercurial ointment daily, or every other day. The treatment must not be pushed to salivation, but should be suspended as soon as a decided improvement in the symptoms begins to manifest itself, and renewed again whenever the improvement seems to flag. It is very important that the strength of the child should be supported by the most nutritious diet possible. It would be wrong, however, to intrust it to a wet-nurse, since this would expose the latter to the danger of infection. That a syphilitic child, although it rarely infects its mother, often does so to its nurse, is simply because the mother is generally syphilitic already, and hence is proof against further contagion.

APPENDIX.

INFECTIOUS DISEASES TRANSMISSIBLE FROM BRUTES TO HUMAN BEINGS.

We shall merely discuss the subjects of glanders and hydrophobia in the following chapters. Regarding the malignant pustule, though this is likewise an infectious malady, communicable from the lower animals to man, yet, as it is more especially a matter of surgical interest, I refer to the text-books of surgery.

CHAPTER I.

HUMAN GLANDERS.—MALLEUS HUMIDUS ET FARCINOSUS.

Etiology.—There is an infectious disease which attacks animals possessing an undivided hoof, especially horses, asses, and mules, and which is called glanders or farcy, according to the locality in which the lesions induced by the virus develop themselves.

The virus of glanders, which is identical with that of farcy, reproduces itself within any organism which it has infected, and the transmission of the virus thus reproduced in one individual to another is the most frequent, and indeed probably the only manner in which the malady is propagated. In other words, glanders is a contagious disease, and probably is a purely contagious one. Its virus, like other contagions, is insusceptible of demonstration under the microscope, or by means of chemical analysis, and we only recognize its presence by its effects. It is contained in the discharge of the farcy-buttons presently to be described, in the flow from the nostrils, the blood, and (*Viborg*) in the excretions, the saliva, urine, and sweat. Whether it is volatile, and capable of pervading the insensible perspiration of the infected person, is doubtful. The fact that the disease may be trans-

mitted at a distance of ten or twelve paces, might be dependent upon the circumstance that particles of nasal secretion might be thrown that distance by a horse, or because the snorting of the animals produces small bubbles of the secretion capable of floating for some time, and of being propelled by the faintest current of air. The glanders may be communicated from the horse, ass, or mule, to various other mammalia. Man is likewise susceptible to it, so that coachmen, grooms, cavalry-soldiers, horse-doctors, and other persons having business among diseased animals, are not unfrequently infected by it. In rare instances, and probably during *post-mortem* sections only, the disease has been transmitted from man to man. The virus of glanders seems to be capable of penetrating the epidermic and epithelial coats, since most cases of infection by it have not been preceded by any wound of the skin.

Anatomical Appearances.—The lesions induced by infection with the poison of glanders consist in peculiar nodules in the mucous membrane of the nose, the lymphatic glands, the skin, the muscles, the lungs, and other organs. At first they are hard, but afterward soften, disintegrate, and form abscesses and ulcers. According to *Virchow*, whose description of glanders we follow, these nodules are the product of a proliferation of cells. In the more recent nodules the cells are young, small, and delicate, and there are many free nuclei; in the older ones the cells are large, distinctly nucleated, lie closely together, and make up almost the entire mass of the tumor. As they progress, the older cells degenerate, and become partially filled with fat globules; they then lose their sharply-defined contour, and break down, so that the nodule finally contains only a mass of detritus, with a few isolated elements. *Virchow* calls attention to the similarity in the progress of development of the farcy and glander nodules, and that of tubercle; but remarks, pointedly, that no inference ought to be drawn from the coincidence of these processes, as the transformation of cells into caseous material is not peculiar to tubercle, and occurs not only in that disease, but also in pus, cancer, and sarcoma.

The nodules which develop upon the nasal mucous membrane of horses, asses, and similar brutes, and which constitute glanders *par excellence*, are of the size of a hemp-seed or pea. The ulcers which result from their bursting are at first solitary, or grouped, but gradually coalesce, imparting to the mucous membrane a peculiar worm-eaten look. The destruction then extends by the development and bursting of new nodules upon the edges of the ulcer, as well as upon its floor, and upon the surrounding parts, and by gangrenous disintegration of large portions of its surface. The ulceration also strikes deeply laying bare the cartilages and bones, which die and are dis-

charged. The eruption of nodules is attended by a nasal catarrh which is very intense around the ulcers, and is at first accompanied by a thin transparent secretion. The discharge subsequently becomes thick, tenacious, and purulent; and when the ulceration is far advanced, it is discolored by the admixture of blood, and becomes acrid and fetid, and contains the *débris* of the necrosed tissues.

In *farcy*, a disease which develops in the skin and lymphatics of horses and similar animals, the tumors are larger than the genuine tubercles of glanders. They contain a greater quantity of caseous matter, are discrete, or else congregate in clusters, or chains and wreaths. After bursting, they form rounded ulcers, with elevated or everted edges, with a foul, irregular bottom, furnishing a profuse ichorous discharge, which often glues the surrounding hairs together, drying up with them into hard crusts.

Both glanders and farcy appear in man. The latter form, however, is the more common, and usually attacks the skin, upon which it generally produces an eruption of tubercles, which are larger and more numerous than those occurring in brutes. The lesions of the nasal mucous membrane are exactly like those found in the horse. Those of the skin, subcutaneous areolar tissue, muscles, and lungs, are purulent rather than caseous; so that in the skin they look like pustules, and in the connective tissue, muscles, and lungs, they bear a great similarity to metastatic abscesses. The lymphatics and their glands are likewise often implicated in the disease in man, and, as in horses, they sometimes produce a chain of farcy-buttons. An inflammation not unfrequently extends from the lymphatics to the neighboring skin, which thus becomes the seat of a malignant erysipelas, with a tendency to gangrene.

Symptoms and Course.—The period of incubation of glanders or farcy is of very variable duration. Where the virus has been implanted upon a wound, the first symptoms generally appear within three or four days; but, when infection occurs where there has been no breach of surface (as when the virus is inhaled), the malady often does not break out for months. The course and magnitude of glanders also differ in the two cases. When the poison acts upon an abrasion, the first symptoms which appear are usually local; the wound inflames; the lymphatics of the part form knotted chains, and their glands swell painfully. The inflammation assumes an erysipelatous character, and is attended by an intense œdema. Blebs form, and pustules, having discolored ichorous contents, and sometimes real gangrenous bullæ, arise upon the skin, and abscesses often develop, or diffuse phlegmonous destruction takes place in parts about the inflamed lymphatics. Sometimes the disease seems to go no further

than this, its effects remaining confined to the lesions about the wound just described, and to a fever of an intensity corresponding to the severity of the case. Signs of a general implication of the system often accompany the above symptoms, however; and indeed they usher in the disease whenever it has not been preceded by a wound.

The term prodromal stage, or stage of invasion, is usually applied to the period during which, although none of the lesions characteristic of glanders or farcy have as yet appeared, still the fever, constitutional disturbance, and certain subjective symptoms, announce a general infection of the system.

Sometimes a single rigor occurs at the beginning of the attack, in others the rigor is repeated several times. The skin grows hot, the thirst augments, the pulse is accelerated, the patient feels depressed and languid, complains of pain in the head, sleeps badly, has no appetite; in brief, exhibits a series of symptoms such as accompany other infectious diseases or local disorders which are accompanied by fever. These manifestations, however, are accompanied by another constant, and somewhat characteristic phenomenon, namely, a violent pain in the joints and muscles. The seat of this pain is usually in the vicinity of the greater articulations. It is generally augmented by motion or pressure, and is sometimes, although not always, attended by moderate swelling of the painful part. Although these articular and muscular pains often cause the disease to be mistaken for rheumatism, yet, when they occur in an individual whose history is suspicious, they may aid us in an early and correct interpretation of the symptoms.

The first stage of the disease lasts for a longer or shorter period (and it may continue for three or four weeks, or more); the symptoms either increase steadily all the time, or else gradually grow milder, so as to subside almost completely when the second or eruptive stage, the stage of localization of the malady, sets in. The aspect of the disease at this period presents many varieties, according as the malady selects one or other tissue as its seat. If it attack the nasal mucous membrane as glanders in the stricter sense of the term, an erysipelatous inflammation makes its appearance upon the exterior of the nose and its vicinity. The nose, the eyelids, and the forehead swell, assuming a dusky redness, and are covered by blebs, the precursors of gangrene. The patient cannot breathe through his nostrils, from which there flows a liquid, at first scanty, thin, and mingled with streaks of blood; afterward foul, bloody, and sanious. If the patient lie upon his back, the discharge flows through the posterior nares into the pharynx, and provokes him to hawk and cough. The discharge causes erosion of the lining of the mouth, soft palate, and tonsils, which are of a deep red hue, and are covered with ulcers and sloughs.

In addition to these manifestations of glanders, or independently of them, when the nose is not attacked, characteristic lesions usually are found upon the skin, which were formerly regarded as a pustulous eruption, until *Virchow* demonstrated that in glanders there was no elevation of the epidermis by an exudation thrown out from the surface of the cutis, but that the skin suffered a circumscribed disintegration, above which the cuticle for some time remained intact. According to *Virchow*, the affected portions of skin at first are intensely reddened, and very small, almost like flea-bites; they then change into papules, and the little elevations afterward assume the aspect of pustules. Beneath their epidermis we find a tolerably thick, yellow liquid, filling up a hole in the corium. The contents of the glanders-pustules, enormous numbers of which often appear upon the body, sometimes grow bloody, and dry up into small blackish or brownish crusts. Besides this scattered eruption, *Virchow* describes clusters of larger "farcy-buttons," which lie deeper in the skin, in the form of large, flat, hard, red tumors, from which the cuticle is finally separated by an effusion of blood, so as to form bluish bullæ. The upper portion of the cutis, which covers these clusters, is also infiltrated by a hæmorrhagic exudation, and afterward breaks down into a pulpy detritus.

The "farcy-buttons" of the subcutaneous areolar tissue and muscles arise either with diffuse inflammatory infiltration of the adjacent parts and all the symptoms of a severe phlegmon, or else small circumscribed, hard, or boggy tumors form, which are quite painless, and then may develop unobserved. Sometimes the contents of the nodules are absorbed; more commonly the skin either ulcerates or sloughs, and great destruction, occasionally extending even to the bones, is the consequence.

Participation of the lungs and bronchial mucous membrane in the disease is announced by shortness of breath, cough, and râles. The deposits are too minute to admit of detection by physical examination.

The larger the number of these symptoms simultaneously presented by the case, and the more rapidly they progress, so much the sooner will the patient succumb with all the signs of the most intense adynamy, and of the so-called typhous or putrid fever, the malady always ending fatally. Death may occur as early as the first day or two, while other cases drag on until the third week.

In chronic glanders and chronic farcy the characteristic lesions of the nasal mucous membrane, muscles, and areolar tissue, are more sparing and less speedy of development than in the acute form, which is of far more frequent occurrence. The skin usually remains intact, while the lungs and bronchial lining are soon affected. Pauses not

unfrequently occur in the eruption, so that the disease seems to have subsided until another cold abscess begins to form or a new eruption breaks out. In chronic glanders and farcy also, the majority of patients finally perish of marasmus, and there are very few instances in which the disease is reported to have ended in recovery.

TREATMENT.—One of the important tasks of a sanitary police should be to diminish the opportunity for the propagation of the virus as much as possible, by the enforcement of strict regulations. Such precautions alone can be productive of certain results, while the utmost vigilance and care will not furnish security, when, either from carelessness or selfishness, the regulations are neglected.

Prevention of constitutional infection, by vigorous destruction of the point of inoculation of the glanders, can only be hoped for immediately after contact of the virus with the wound. The remedies proposed for the cure of pronounced glanders, calomel in large doses, iodine, Fowler's solution, injections of a strong solution of creosote into the nostrils, and the cold-water cure, are all productive of but little benefit, according to our present experience. A treatment of the symptoms, in which local disturbance, fever, and the strength of the patient, are taken into account, is the one most to be commended.

CHAPTER II.

HYDROPHOBIA—LYSSA—RABIES.

ETIOLOGY.—The difficult question about the nature of rabies and its proper position in the nosological system remains unsettled. The fact that the poison, the absorption of which induces the disease, is reproduced in the body of the patient, goes far toward proving that it is infectious. This view is also favored by the symptoms occurring among certain classes of animals. The two chief forms of rabies in dogs, the quiet and raging, remind us of the febris nervosa stupida and febris nervosa versatilis of old authors. On the other hand, there are certain objections to classing lyssa among typhus fevers, the acute exanthemata, and other acute infectious diseases. I would especially call attention to the variable duration of the period of incubation in different persons. We do not certainly know the length of time between the action of the contagion and the outbreak of the malady in all infectious diseases, but we suppose that the difference of this interval is only slight in different persons. In lyssa the period of incubation varies weeks and months, even if we do not consider

extreme cases. Moreover, there is an essential difference between the character of the fever in this and other acute infectious diseases.

In lyssa, among the symptoms are some nervous ones, slightly analogous to the neuralgias which occasionally occur, instead of the fever in malarial infection. Lyssa humana most resembles tetanus, although I cannot understand how it is possible to deny the occurrence of lyssa as an independent disease, and to identify it with tetanus. In the well-observed cases, neither continued tension of the spinal muscles nor pain in the contracted muscles has been mentioned, while neither is ever wanting in true tetanus. The difference between lyssa and tetanus is, that the spasms, which may be termed tetanic, only occur in some paroxysms, and are limited to certain nerves, and more particularly to those which are by no means chiefly affected in traumatic or rheumatic tetanus. When *Romberg* terms lyssa a toxoneurosis, he says all that we actually know of the nature of the disease.

Without the experience that the poison is reproduced in the body of the patient, we might most reasonably suppose that a toxic neuritis, or a toxic neuropathia, spread from a peripheral nerve, on which the poison had worked, to the medulla oblongata and cerebrum; and, since in many cases changes in the wound or in the cicatrices precede the outbreak of the malady, and the time between these changes and the outbreak is very short, it would be most probable that the poison had remained latent at the seat of injury, but afterward had rapidly spread in a centripetal direction. This latency would most remind us of that of syphilis between the different outbreaks of the disease. However, all this reasoning lacks a solid foundation.

Lyssa, or rabies, in its stricter sense, is a disease which originates in animals of the dog kind, and which indisputably is propagated, in most instances, by infection of one animal by another. We shall not discuss the question whether this be its only mode of propagation, or whether, under certain circumstances, it may develop spontaneously. The rabies of other races of animals and the hydrophobia of human beings are purely contagious maladies. This poison, which exists in the saliva, blood, and perhaps in other fluids of the body, is not volatile, but fixed in character; it cannot pass through the epidermis, and hence does no mischief, unless brought in contact with an ulcerated or excoriated surface.

The most common cause of human rabies is the bite of a mad dog. More rarely the disease originates in the bite of some other rabid animal, such as the wolf, the fox, the cat, or the ox. The possibility of infecting a healthy man by the bite of a man suffering from hydrophobia has not yet been proved positively; but the inoculation of the disease from human beings to brutes has repeatedly been successful

Bites of rabid animals upon bare portions of the body are far more dangerous than if the part bitten be covered by the clothing; as, in the latter case, the poisonous saliva is not so readily conveyed to the wound, being wiped off from the teeth by the clothing. For the same reasons, there is no danger from the licking, or otherwise moistening, of the sound skin by the saliva, blood, or other fluids of a rabid animal, unless there should happen to be an abrasion or fissure upon its surface.

The virus, when implanted upon an excoriation of the skin, does not lead to hydrophobia unless there be a certain degree of predisposition to the disease. Inoculations of the saliva of rabid animals, as practised by *Hertwig*, succeeded only in twenty-three per cent. of the animals operated upon, seventy-seven escaping; and, according to *Faber's* statistics, out of one hundred and forty-five persons bitten by rabid animals, in Würtemberg, only twenty-eight had hydrophobia.

Anatomical Appearances.—No lesions characteristic of the disease are found in the bodies of those who have died of hydrophobia. The most common conditions consist in intense rigor mortis, extensive *post-mortem* hypostasis, early putrefaction (so that, soon after death, blebs full of gas begin to arise), intense staining of the endocardium and walls of the vessels, hyperæmia and serous exudation in the brain and its membranes, in the spinal marrow, in some of the sympathetic ganglia and nerves; hyperæmia and swelling of the mouth and fauces, both of which contain a collection of tenacious mucus; hypostasis and œdema of the posterior parts of the lungs; engorgement of the walls of the stomach, and great abdominal glands. All these lesions, especially the injection of the nervous centres and nerves, upon which, at times, great stress has been laid as explanatory of the nature of the disease, are not constant, and, for the most part, seem to arise just prior to dissolution, in consequence of the disturbance suffered by the functions of respiration and circulation during the attacks presently to be described. In the cases which I have seen, autopsy showed a decided swelling of the tonsils and follicular glands at the root of the tongue and the posterior wall of the pharynx, exactly corresponding with *Virchow's* observations.

Symptoms and Course.—Most cases of lyssa that have been well observed and described closely resemble each other. One that I have seen corresponds closely to the dreadful picture which *Romberg* drew from his own and other cases. As it is universally assumed that morbid processes due to the action of a specific poison run their course with symptoms which only vary through personal idiosyncrasy and the variable intensity with which the poison has acted, those reports of lyssa humana differing from our description, in which the characteristic symptoms and their peculiar sequence are not

mentioned or really did not occur, must arouse the suspicion that they were badly observed, or that there was an error of diagnosis.

Opinions differ as to the length of its period of incubation. The statements that hydrophobia has made its appearance twenty or thirty years after the bite of a rabid animal, as well as those according to which the disease has broken out as early as the second or third day, are probably dependent upon imperfect observation. The shortest term of incubation appears to be about eight or ten days; the longest, twelve or thirteen months. In the majority of instances, the malady breaks out in about forty days after the reception of the bite. The reasons for this inequality of the period of incubation are obscure. The assertions of *Marochetti*, who claims that, during the incubative stage, vesicles form beneath the tongue, and that, by destroying these vesicles, the outbreak of the disease can be averted, have not been substantiated. But there are numerous instances in which, toward the end of the stage of incubation, and a day or two before the onset of the malady, peculiar alterations have been observed in the wound or its scar, for the wound has generally healed by this time. The bite assumes a livid color, grows painful, and discharges a yellow ichor. The scar, which has generally soon formed without remarkable symptoms, grows bluish red, swells, and sometimes breaks out afresh. The patient also complains of painful sensations, shooting centripetally from the wound or scar, or of a sense of numbness in the bitten member. These disturbances at the point of entry of the virus are very often wanting.

The first or prodromic stage of the disease is marked by a peculiar depression of the patient's spirits, amounting to an acute melancholy, and which has caused the term stadium melancholicum to be applied to this period of the disorder. The patient seeks solitude, is timid and apprehensive, and either sits motionless and plunged in deep abstraction, or else is unable to rest at all. Some complain of an indefinite feeling of dread and oppression, and sigh repeatedly without any reason for so doing. Some are preoccupied with sad forebodings, or, if aware of their perilous condition, are incessantly tormented by dread of the onset of the malady. Sleep is restless and broken by frightful dreams. The precursory signs of the spasmodic disturbance of respiration, afterward to attain so terrible an intensity, soon supervene. The patient complains of pressure in the præcordium, draws profound, sighing inspirations, the diaphragm is depressed, the epigastrium bulges, and the shoulders are drawn upward. This spasmodic breathing is the first token of the tonic spasm of the muscles of inspiration which causes such frightful torments in the second stage of the disease.

The prodromic term having lasted two or three days, the second or furious stage begins. Its onset is marked by a fit of choking, suddenly induced by an attempt to drink, which renders the patient incapable of swallowing a drop.

The moment the fluid enters the mouth and the motion of swallowing is made, spasmodic inspiratory motions begin; the thorax rises interruptedly, and remains in the position of deepest inspiration for ten or twenty seconds. During this time the features betray anxiety and terror, the eyes protrude, head and shoulders are thrown back, then comes an expiration, with which the attack passes off. I have satisfied myself that the inspiratory muscles, as well as those of the pharynx, are implicated in these seizures. This combination of spasmodic contractions in both of these sets of muscles is a frequent symptom; it always appears in the straining preceding vomiting, as well as in the retching following irritation of the pharynx by the finger, etc. Retching is always accompanied by a feeling of suffocation, from the contraction of the inspiratory muscles complicating the spasm of the pharynx; and we are justified in giving the name of retching-fits to these spasms, which form the pathognomonic symptom of lyssa, and are given in every well-recorded case.

The dread of water is entirely due to the dreadful experience of the patient on trying to drink. This was so, not only in my case, where the patient, an energetic and courageous man, voluntarily made repeated attempts to drink, before he rejected with terror any suggestion of the kind, but it is the same in all cases of undoubted lyssa that have been well observed and accurately described. Reports of cases where hydrophobia is given as a primary symptom are worthy of no credence. It is a curious fact that, at first, the act of swallowing solid food is not followed by spasms. Even a draught of air on the skin, or touching any thing cold, sudden irritation of the eye by dazzling light, even sudden mental excitement or surprises, may induce attacks. According to my observation, the pharyngeal muscles do not participate in the reflex spasms induced by irritating other parts, as the mouth or palate. On suddenly arousing my patient, he opened his mouth widely, threw the head back, the thorax was raised as in full inspiration, the epigastrium became prominent, but there was no actual retching. At the height of the disease, it looked as if attacks occurred from time to time, even without cause; but I think that even these apparently spontaneous attacks must be regarded as reflex spasms, and be referred to collections of tough mucus in the pharynx or to the trickling into it of saliva. This supposition is based on the haste and *abandon* with which the patients eject saliva and mucus,

and their attempts to introduce the finger far into the throat, for the purpose of removing mucus and sputa.

Various authors name tetanic or epileptiform spasms among the symptoms of lyssa; but, on careful examination, I have not been able to discover a case where the detailed description of the spasms fully convinced me that they were tetanic or eclamptic. It is nowhere stated that the muscles of the back were tense, except during the attacks, or that consciousness was lost during the general convulsions. In my patient, there was also opisthotonos—he threw his hands and feet about, and pitched around, so that he frequently fell on the floor. But these symptoms reminded one far more of hysterical spasms, or of the actions of a tortured, despairing man.

The above symptoms are soon accompanied by attacks of boundless rage, in which the patients are hard to manage, destroy all that comes in their way, strike, kick, scratch, and bite, if held fast—and, not unfrequently, kill themselves, if they are carelessly watched.

The biting, inarticulate howling, and barking sounds, are not made more frequently by a hydrophobic patient than by another madman in the maniacal stage of chronic cerebral disease.* The patient often warns his attendants between the fits, which seldom last longer than a quarter or half an hour, and begs pardon for his misbehavior toward them, and sets his worldly affairs in order, in perfect consciousness of the near approach of his end. The paroxysms of madness and convulsions, having steadily grown more frequent for two or three days, now begin to diminish in violence as the patient loses strength. Rarely, it happens that death occurs at the height of the malady, during a severe and long-continued choking-fit. The exhaustion and collapse usually augment from hour to hour; the voice grows hoarse and feeble, the respiration shallow, the pulse small, irregular, and very frequent, and death ensues with the signs of a general paralysis, which is sometimes preceded by a deceptive amelioration of the symptoms.

It might be supposed that the attacks of madness occurring in lyssa were simply a result of the despair that would affect even a person not having this disease, if he suffered from retching at short intervals for a day or two. I once attended a patient suffering from severe pharyngitis, who, when I asked him to try to drink, hurled the glass from him, and acted like a madman.

We find something like this, too, in patients with croup or œdema glottidis. The fact, also, that sometimes patients of very temperate

* *Romberg* says that a great inclination to bite, along with the absence of characteristic reflex spasms, is one of the diagnostic points between true lyssa and those hypochondriacal and maniacal conditions that fear of the disease not unfrequently develops in persons that have been bitten. This state might be termed lyssophobia.

and resigned natures do not become maniacal would also favor this view. But there are some objections to it, especially the fact that, even in persons the most resigned, the absence of mania is one of the rarest exceptions, as well as the excessive height that the madness usually reaches in lyssa patients. It is certainly more probable that the madness in lyssa is not due to moral grounds, but is caused by a propagation of the excessively increased morbid excitability of the motor-central apparatus of the pharyngeal and respiratory nerves to the central organs of the psychical functions. The symptoms of the mania have many analogies to reflex spasms. Trifling mental excitement causes severe outbreaks, violent motions, and excited actions in manaical patients, just as slight irritation of the skin causes severe reflex spasms in patients with tetanus.

Treatment.—We shall only make the following brief remarks about the sanitary measures by which the state should try to protect its subjects. The only prophylaxis is by confinement of dogs. No attention should be paid to the lovers of dogs. Any one who expends sympathy on the "poor dog," and petitions against his being tied up, or wearing a muzzle, should be made to watch a patient with hydrophobia for half an hour, and he would soon be cured. Most muzzles that dogs wear do not prevent their biting, and they are only protective when they do so. It is very important not to kill dogs suspected of madness, but to shut them up and observe them closely. Death, which soon takes place spontaneously, and the symptoms under which it occurs, are far more important in determining that the dog was mad, than are the results of autopsy; for, from the latter, especially if the dog was killed by violence, it cannot be said with certainty whether the dog was mad or not, and harm has been done by such unreliable, demonstrative assertions. The most we can say is that negative results of an autopsy, where no changes have been found in any organ of a dog suspected of rabies, to explain the symptoms and death, go to prove the probability of the diagnosis—especially if we find hair, straw, rubbish, etc., in the stomach; for, while a healthy dog would not be apt to swallow them, a mad one would.

If a man has been bitten by a dog suspected of rabies, prophylaxis requires a destruction of the spot with which the poison may have come in contact; and every dog that has bitten without provocation, or without previous inclination to bite, should be suspected.

The object of prophylactic treatment should be to destroy the portions of skin which have come in contact with the virus. It consists in excision of the wound, and in the vigorous cauterization of the part

with the hot iron, or with a deliquescent caustic, such as caustic potash, butter of antimony, and the like. It is also advisable not to permit the sore, resulting from detachment of the eschar thus made, to heal too rapidly, but, if practicable, to keep it suppurating for months. These measures are all the more likely to be successful, the earlier they are applied, but they should never be neglected, even though weeks have elapsed since the infliction of the bite.

As a further precautionary treatment, the exhibition of mercury to the point of salivation, large doses of belladonna, and a series of other so-called antilyssa, are employed; but it is very questionable whether the disease has ever been averted by the use of any of these articles, and whether the cases in which hydrophobia has not followed upon the bite after the use of anagallis arvensis, gentiana cruciata, rosa canina, genista luteo-tinctoria, cantharides, or may-worm, did not belong to the seventy-seven per cent. of subjects in whom the predisposition to rabies did not exist, and without which hydrophobia never occurs. Inasmuch as, after the establishment of the disease, these measures do not produce even the slightest palliative effect, their prophylactic virtue is more than doubtful; hence, I by no means regard it necessary to subject a man, who has been bitten by a mad dog, to the baneful influence of mercury, although I should not hesitate to resort to any procedure, be it never so severe or dangerous to the life of the patient, if, according to actual experience, any benefit were to be expected from it.

If the disease has broken out, there is little hope of curing it, or even of alleviating the sufferings of the patient, for hitherto there has been no accurately observed case of lyssa having any other termination than a painful death. Even the waiting and nursing are very difficult, and should only be trusted to persons who are humane as well as fearless and energetic. Every thing that can induce return of the spasms, and, later, of the paroxysms of madness, should be carefully avoided. To relieve the thirst, small enemata of cold water may be given from time to time. In the case I saw, these were well borne and quickly absorbed. In recent cases, where the patient is young and vigorous, we may bleed, as in some cases this has proved of undoubted benefit. If the patient will bear it, we may also let him inhale chloroform from time to time, and give subcutaneous injections of a strong solution of morphine. The favorable results said to have been attained in some cases of tetanus, by the use of curare, decided me to try it in a case of lyssa under my care. At first $\frac{1}{10}$th, later $\frac{1}{5}$th of a grain of carefully-tested curare was subcutaneously injected at intervals of three to four hours. Distrustful as we were about the result, this treatment still seemed of palliative benefit, and seemed

to afford greater relief than the morphine injections. The patient repeatedly prayed for the continuance of the curare injections, as he was firmly persuaded that they relieved him. This experience is enough to make me urge injections of curare in any case of lyssa humana; and, as nothing can be lost by it, to give it even more boldly than we did.

SECTION III.

GENERAL DISORDERS OF NUTRITION WHICH DO NOT DEPEND UPON INFECTION.

CHAPTER I.

CHLOROSIS.

Etiology.—The subjects of anæmia and hydræmia belong rather to the province of general pathology, since such anomalies of the quantity and quality of the blood never arise as independent affections, but always appear as an accompaniment or consequence of other diseases. The state of the blood which gives rise to the condition known as chlorosis differs from that which induces hydræmia. In chlorosis, only the cellular elements of the blood are diminished in number, while its quantum of serum, albumen, and saline constituents, is generally normal. In hydræmia, on the other hand, not only is the blood poor in cellular elements, but its serum is deficient in albumen, and it probably is overcharged with salts. A chlorotic state of the blood (oligocythæmia, *Vogel*) often develops with a certain degree of independence; or, to speak more accurately, it frequently arises without our being able to perceive any morbid condition which, by augmenting the consumption of the blood, or by diminishing its production, might induce chlorosis, and which generally can be discovered in anæmia and hydræmia. In the present chapter it is the former class of cases alone with which we have to do, and we shall not notice those rare instances in which oligocythæmia develops as a symptom in certain diseases.

In females between the ages of fourteen and twenty-four, chlorosis is one of the most common of disorders. It is very natural, then, to ascribe the disease to the effects of those processes which are going on in the bodies of young girls at the period of puberty; but we are still ignorant as to what physiological connection exists between the two conditions. We know equally little of the causes

which favor the occurrence of chlorosis at the time of puberty; for, although it is not improbable that the development of this affection is often encouraged by a want of fresh air and exercise, by improper nourishment, mental excitement, improper reading, masturbation, or by a generally unhealthy mode of life, yet the disease arises often enough under conditions precisely the opposite of these, in girls who work all day in the open air, who are well fed, do not read novels, and are not addicted to any secret vice. I may add that, according to my observation, obstinate chlorosis attacks all young girls without exception, in whom the menses have appeared in the twelfth or thirteenth year, and before the development of the breasts and pubes.

Far more rarely chlorosis—oligæmia without assignable cause—appears in children and in pregnant women, and sometimes even in males. The number of instances of chlorosis in this class is very limited.

Anatomical Appearances.—The lesions characteristic of chlorosis lie mainly in the blood, which, with *Virchow*, we may regard as a tissue consisting of cells with a liquid intercellular substance. As has been said already, this intercellular substance, the serum, does not present any constant anomaly. Its composition generally is normal; more rarely there is a diminution of its albumen. In other cases, again, the amount of albumen of the blood-serum seems to be increased, so that, besides the oligocythæmia, there is hyperalbuminosis. In the first two instances, the whole volume of the blood is probably reduced, while in the latter the possibility cannot be denied that, in spite of the diminished number of the blood-corpuscles, the absolute bulk of the blood is augmented, adding a serous plethora to the oligocythæmia.

In pronounced chlorosis, the disease in the red-blood corpuscles may be so great that a thousand parts of blood may contain but sixty or forty parts of dried blood-cells, instead of the normal average of one hundred and thirty parts. Upon the autopsy of a chlorotic person, who has died of intercurrent disease, the viscera are all found to be remarkably pale. In some cases, the signs of simple fatty degeneration are found in the tunica intima of the great vessels.[1]

Symptoms and Course.—The most striking symptom of chlorosis consists in the pallor of the skin and visible mucous membranes. When the skin contains but little pigment, as is the case in blondes, the surface of the body is of a pure white; while, when the pigmentation is intense, as in black-haired persons, it is more of a dirty-gray or yellowish hue. The pallor is often most distinctly pronounced upon the ears, while in the mucous membranes the loss of color is most remarkable in the conjunctiva and the gums. The reason for this blanching of the complexion is manifest. The number of red-blood corpuscles

upon which the blood itself depends for its redness, and the tissues through which it circulates for their tints, is reduced one-half, or one-third, or even lower. The exceptions to this rule, namely, the occasional instances of chlorosis in which the cheeks retain their redness, are easy of explanation, when we come to know that the color of the blood is but one of the causes upon which the tint of the cheek depends; another consisting in the filling and distention of the capillaries. Just as there are persons whose cheeks are pale, in spite of the deep hue of their blood, because their capillaries are inadequately filled, so there are other chlorotic persons who, notwithstanding that their blood is pale, always have red cheeks, because their capillaries are filled to bursting, and are varicose. The transient flush observed in almost all chlorotic persons when excited or heated, and to which we shall refer hereafter, is also to be ascribed to a momentary distention of the capillaries with blood.

The fat in the subcutaneous areolar tissue of chlorotic persons is often normal in amount, and is sometimes even in a state of exuberant development. This condition furnishes an important distinctive mark between chlorosis and the chronic anæmia which often manifests itself in latent consumption, and in other concealed disorders which affect the quality of the blood. In a symptomatic anæmia or hydræmia of this kind, which is frequently confounded with chlorosis, the subcutaneous fat soon vanishes as the color of the skin fades. Œdematous effusion into the subcutaneous cellular tissue is rare in this disease. Hence a moderate paleness of the surface, accompanied by œdema of the feet, warrants the suspicion of hydræmia and not of chlorosis; and conversely, when the lips, cheeks, and ears are white as wax, and yet there is no sign of œdema, it is probable that the serum of the blood retains its normal composition, and that the case is a pure oligocythæmia and not a hydræmia.

Chlorosis is always accompanied by more or less shortness of breath, owing to a diminution in the number, sometimes amounting to one-half or more, of the bodies which take up oxygen, and give out carbonic acid. The normal number of respiratory acts is insufficient to supply oxygen to the lungs in quantity adequate to the wants of the system. Every bodily effort, by increasing the consumption of tissue, and augmenting the production of carbonic acid, aggravates the dyspnœa, and multiplies the patient's breathings to a distressing degree. Hence patients with pronounced chlorosis scarcely ever fail to complain that they "get out of breath" when they walk fast, or mount the stairs. The diminution in the interchange of gases, and especially the impediment to oxygenation of the blood, also accounts for a second series of symptoms peculiar to chlorosis. The strength of the muscles,

which, to be vigorous, calls for a supply of well-oxygenated blood, is greatly reduced. The patient is easily fatigued, and complains of a sense of weight in his limbs, and not unfrequently (pseudo-rheumatic) muscular pains are induced by the slightest effort, such as only are felt by healthy folk after unusual and excessive exertion. Important as it is to the normal function of the muscles that a free supply of well-oxygenated blood should circulate within them, this is equally essential to the proper action of the nerves. In treating of the neuroses, in almost every instance, we have referred to the state of the blood in chlorosis as an important point in their etiology. Hence, neuralgia is extremely common among chlorotic patients, that of the trigeminus being especially frequent among the peripheral neuralgias, while in the internal organs cardialgia is more common. Anæsthesia, convulsions, and palsy, are rarer, and are seldom observed excepting when (as sometimes happens) hysteria develops during the disease. There are nearly always signs of hysteria in chlorosis, which consist of general bodily and mental hyperæsthesia, a troubled, irritable temper, a disposition to weep, perverted appetite (for instance, a craving for coffee-beans, slate-pencils, etc.).

The organs of circulation present numerous deviations from the normal. The patients nearly all complain of palpitation. I have already declared that this symptom is not altogether due to an augmented or accelerated action of the heart, being also in some degree dependent upon a general hyperæsthesia, whereby the impulse of that organ is rendered distressingly perceptible, whereas it is not felt at all by most persons, even when its beat is enormously intensified by hypertrophy (Vol. I.). Upon auscultation of the chest, over the heart and great vessels, we hear the blowing sounds so often alluded to, and which are known as "blood murmurs," to distinguish them from the morbid murmurs depending upon alteration of the structure of the heart. This name is inappropriate, however, as the sounds are not caused directly by the altered condition of the blood. They most probably arise from an abnormal tension of the valves and arterial walls.

Far more frequently, upon listening over the jugular vein, a peculiar humming, singing murmur is heard, known as the "Nonnengeräusch," or "bruit de diable." The name is derived from the sound of the humming-top, called "Brummkreisel" in North Germany, and "Nonne" (nun) in other places; while in France it is called the 'diable." The sound is more intense upon the right side than upon the left, and subsides when the patient assumes a horizontal attitude or draws a forced breath. The mode of origin of the "bruit de diable" seems to be as follows: The lower part of the internal jugular veins

lying behind the sterno-clavicular articulation is attached upon all sides, so that it cannot collapse, like other veins, when the stream of blood is reduced in volume. When the jet of blood which enters this large space is too small, it can only fill it by forming an eddy. This whirling current throws the walls of the veins into sonorous vibration. If the neck be turned to one side so as to compress the jugular of the opposite side between the omohyoid muscle and the cervical fascia, the bruit de diable becomes audible in the majority even of full-blooded, healthy persons. When the sound is perceptible without such a turning of the neck, it may always be inferred that the patient's blood is impoverished and that his veins are scantily filled. The excessive nervous irritability arising from inadequate oxygenation of the blood also involves the vasomotor nerves; whence the sudden flushing and paling of chlorotic persons.

Besides the cardialgia already alluded to, other serious disorders of the digestive system arise in chlorosis, which, unless watched with patience and attention, might give rise to dangerous mistakes. The appetite is nearly always diminished; after eating, there is a sense of pressure and fulness in the epigastrium, with acid eructations and other symptoms of dyspepsia, which, in most patients, depend on "atonic weakness of digestion" (Vol. I.), and generally disappear as the state of the blood improves. Affections such as these are not dangerous, and nearly always get well under proper treatment; unfortunately, however, the chronic ulcer of the stomach is also a very common occurrence in chlorosis, and often develops undetected. In treatment of a chlorotic patient, it must never be forgotten that the cardialgia and dyspepsia may be dependent upon this grave lesion of the stomach, and all suitable means must be invoked to aid us to a correct diagnosis. Neglect of such precautions may terminate in an unexpected and tragic *dénouement* by hæmatemesis, or even by perforation.

The urine of a chlorotic patient, unless there be intercurrent fever, is remarkably limpid and light. The lowness of its specific gravity is probably owing to the small amount of urea which it contains, and its light color to a deficience in coloring matter. Reduction in the supply of oxygen sufficiently accounts for the decrease in the destructive assimilation, and consequently for the diminution of the urea in the urine. We know but little regarding the formation of its coloring matter, but it can scarcely be doubted that it is derived from that of the blood; hence, when the number of the blood-corpuscles, which contain the red matter, is diminished, it is not surprising, cæteris paribus, that the coloring of the urine should decrease.

The sexual function nearly always suffers derangement, generally in the form of amenorrhœa, more rarely in the form of excessive men-

struation or of dysmenorrhœa (Vol. II.). When amenorrhœa occurs, it is almost always because the ovules do not mature; for, generally, all the other signs which attend ripening and expulsion of the ovum are absent, besides the bleeding. By the laity, and even by some physicians, amenorrhœa is supposed to be a constant symptom of chlorosis, an error which may sometimes be productive of the utmost mischief, by inducing the premature suspension of remedies still urgently required, on account of the presence or appearance of the menses. Indeed, owing to this erroneous idea, that chlorosis is always attended by amenorrhœa, it not unfrequently happens that chlorotic girls, suffering from dysmenorrhœa or menorrhagia, and complaining of headache and palpitation, are supposed to be "too full-blooded," and are therefore purged and bled. Finally, in many chlorotic patients, besides the irregular menstruation, there is a catarrh of the uterus and vagina (Vol. II., pp. 123, 171).

The course of chlorosis, unless cut short by proper medication, is almost always slow and tedious. This disease, if not detected, or if treated homœopathically, often lasts for months, and sometimes for many years. It generally terminates in recovery, although, in very young persons, one or more relapses are apt to occur. More rarely, chlorosis passes over into other disorders, such as tuberculosis. It never endangers life, excepting through its complications, particularly the very common one of chronic ulcer of the stomach. The stories about acute febrile chlorosis terminating in death are the result of erroneous diagnosis; although it is true that unimportant febrile diseases, when they attack chlorotic patients, are often accompanied by typhoid symptoms and assume a malignant type.

Treatment.—Attempts to fulfil the causal indication in chlorosis are very seldom followed by benefit, a proof that the causes to which the disease is generally ascribed are not relevant ones. Chlorotic girls are often restored to health in a few weeks, by the fulfilment of the indications from the disease itself, although they had been long and carefully, though fruitlessly, protected from pernicious agencies, had passed months in the country, lived on the best of food, exercised daily, and taken a course of German or Swedish gymnastics without bringing color to their lips and cheeks, relief to their shortness of breath, or improvement to their irritable and fretful temper. The reputation which dietetic treatment enjoys, as a remedy for chlorosis, is mainly attributable to the circumstance that not only does pure oligocythæmia bear this name, but it also is given to a great variety of anæmic conditions, in which such treatment is indispensable, while the use of iron is but of secondary importance. Experience teaches daily that brilliant cures of pure chlorosis may be made under the most unfavorable external conditions.

The indications from the disease itself demand the exhibition of iron. If any medicine ever deserved the name of a specific, iron does, as a remedy in this disease. The surer our diagnosis, so much the more certain may we be of succeeding; and when, contrary to our expectations, we fail of success, there will be reason for suspecting that the case is not simple chlorosis, but an anæmia depending upon some other unperceived or unrecognizable disorder. The progress of a chlorosis, which, developing in a young girl at the period of puberty, has defied all treatment, often proves it to have been but the initiatory stage of a tuberculosis, or the anæmia consequent upon a chronic gastric ulcer. We are ignorant of the manner in which the iron improves the defective state of the blood. The existing red-blood corpuscles are not wanting in iron, but there is a deficiency of the red corpuscles themselves, into whose chemical composition iron enters to a slight extent. Perhaps the iron stimulates the activity of the region where the red corpuscles are formed, or perhaps it regulates the digestion, and thus promotes the supply of material for the formation of blood-corpuscles. But there is no sound foundation for any of these hypotheses, the number of which might be materially increased.

Opinions vary greatly as to the proper form and dose of iron. Iron-filings are much prized by certain well-known and fortunate practitioners; others, equally celebrated and successful, prescribe one or other of its salts, to the exclusion of all the rest, claiming its effects to be more certain, and better borne. Others, again, begin treatment with the "mildest" of the ferruginous preparations, as the mallate or lactate of iron, proceeding thence to the "stronger" ones, the chloride and sulphate, winding up the treatment with the filings. Besides the limatura ferri, which may be given in three to six grains as a dose, generally with an addition of powdered cinnamon, the tinct. ferri pomat. (gtt. xv–xxx), the lactate of iron (gr. ij–v), the ferri carb. saccharat. (gr. iv–x), the tinct. ferri chlor. (gtt. x–xxx), ferri sulph. (gr. j–iv), are the preparations of iron in most common use. From the estimate in which these various preparations are held by different established authorities, and from the fact that nearly every practitioner has his "pet article," which he uses in the majority of cases, it may be inferred: 1. That the efficacy of iron in chlorosis does not depend upon the form in which it is administered. 2. That nearly all ferruginous preparations are well borne in chlorosis. 3. That special indications for the exhibition of one or other of them cannot be laid down. For more than twenty years I have used *Blaud's* pills almost exclusively in chlorosis, and have witnessed such brilliant results from them in a large number of cases, that I have never found any opportunity to experiment with other articles. Instead of the forty-eight huge boli,

according to *Blaud's* original prescription (℞ Ferri sulp. pulv., potass. carb. puræ, āā ʒ ss, tragacanth. q. s. u. f. pil.), I have ninety-six pills made. Nor am I so timid in increasing the dose according to *Blaud's* formula, but order three pills thrice daily, and sometimes four or five if they are well borne, which they almost always are. Three boxes of *Blaud's* pills nearly always suffice to cure the most persistent chlorosis. At Magdeburg and Greifswald I often had to send my recipe for the pills to a great distance, my good fortune in the treatment of chlorosis—to which, by-the-by, I owe the rapid growth of my practice—having given me a great reputation as the possessor of a sovereign remedy against that disease.

I do not suppose that *Blaud's* pills excel all other ferruginous preparations in virtue; indeed, I have repeatedly satisfied myself that colleagues who made use of other recipes obtained equal results, provided only that they gave doses of equal size; but I also believe that the efficacy of *Blaud's* pills cannot be surpassed, simply because they can be administered in very large doses without distressing the patient. I had an opportunity to test the truth of this some ten years ago. I was asked for my recipe by one of my colleagues, and at first referred him to the Canstatt text-book, which contained the well-known formula for *Blaud's* boli; but, as he did not have that book, I told him the formula, adding, that I began with three boli, raising the dose one bolus every three days. Some time afterward my colleague took occasion to thank me, extolling the excellent effect of the medicine, and stating how well it had been borne by the patient. As it turned out, however, he had misunderstood me, and, instead of raising the dose every three days, had added one bolus at every dose.

It has been maintained that the treatment of chlorosis does not require large quantities of iron, because the amount of it taken up by the blood is very small, and since, even when small doses are given, a large portion of the metal always passes off in the stools as superfluous, and further, because the efficacy of the chalybeate springs in this disease proves that a minimum of iron will suffice. Without going into groundless theoretical discussions, I will admit that the disease will also recover (although somewhat slowly) under the use of smaller doses of iron, and may be cured by chalybeate waters; but the number of patients who have recovered in my practice through the employment of *Blaud's* pills, and under that of my colleagues under the use of carbonate of iron (pil. ferri carb.), or of lactate of iron in large doses, after they had taken small doses of tincture of iron and wine of iron for years without positive effect, and had repeatedly visited Pyrmont and Driburg, is large enough to warrant the assertion that we shall cure chlorosis most speedily and surely by means of those ferruginous

preparations which can be tolerated in the largest doses, and of these *Blaud's* pills stand highest on the list.

A very common error in the treatment of this affection consists in the attempt to relieve the erethism, digestive disturbance, and other troubles due to the state of the blood, by means of mineral acids, bitters, and other medicines; while iron, from which alone, or at least from which the greatest benefit is to be anticipated, is neglected. Such preparatory treatment almost always retards the recovery unnecessarily.

I will finally observe that, as long as my chlorotic patients continue languid, indisposed to exertion, and void of appetite, I do not urge them to walk or to eat, generally to their great satisfaction; but I make them promise that they will exercise assiduously as soon as they feel the strength and inclination to walk, and that they will take food freely whenever they gain an appetite to eat, the acquirement of which is seldom long withheld. I have already said that relapses of chlorosis cannot be averted, especially when it sets in at the commencement of the period of evolution; hence I always take the precaution of suggesting the possibility or even the probability of a relapse, and have often found that the patient and her relatives do not take the prospect much to heart, when I assure them that the second attack will be as speedily curable as the first.

Although, as I have said, I consider the chalybeate springs superfluous, and far less effective than iron given in large doses, yet, when a convalescent from chlorosis fears a relapse, I would recommend the springs of Pyrmont, Driburg, Cudowa, Altwasser, St. Moritz, in Switzerland, and Imnau, in Swabia.

CHAPTER II.

SCORBUTUS—SCURVY.

Etiology.—The abnormal changes in the quality of the blood to which chlorosis is due can be detected by microscopic and chemical investigation; these aids fail us in our researches upon scurvy. It has been stated, indeed, that, in scorbutic blood, the fibrin is diminished, or has lost its coagulability; or that the salts of soda are abnormally increased, while those of potash are diminished; but, after repeated investigation, the truth of none of these statements has been established. Nevertheless, the commonly adopted opinion, that scurvy is a disease dependent upon a derangement in the composition of the blood, is probably correct. Independently of the fact that the disease arises under conditions unfavorable to a normal composition of the blood, we

shall see, while discussing its symptoms, that these depend upon a disease of the capillary walls, extending throughout so large a portion of the system that we must infer that the starting-point of the malady is an imperfect state of nutrition of the capillary walls resulting from improper nourishment.

With regard to the etiology of scurvy, we shall limit our remarks to a statement of the conditions which are found to favor the development of the disease, since, until we know in what the scorbutic state of the blood consists, all theoretical reasoning upon its connection with these pernicious conditions is idle. Scurvy is so common an occurrence during long voyages at sea (sea-scurvy), where the crew live almost entirely upon hard bread and salt meat, and are entirely deprived of potatoes and fresh vegetables, that the disease has been attributed sometimes to the over-supply of salt in the food, sometimes to want of fresh meat and vegetables, and sometimes again to both causes combined. The theory, that in scorbutic blood there is an undue preponderance of the salts of soda over the salts of potash, seems to find support from the frequence of this affection among seamen who are deprived of vegetables, while profusely supplied with salt. Nevertheless, although it cannot be denied that the food of sea-faring people during long voyages furnishes one of the conditions which favor the occurrence of scurvy, yet this is by no means to be regarded as the sole cause of the disease. Scurvy has been known to break out early, and with great virulence, in ships where the crew have been greatly exposed to cold, particularly to moist cold, as well as in ships which have been detained by protracted calms at the equator. It has also been observed that immoderate fatigue and a despondent, melancholy state of mind favor the development of the disease, while a crew who work moderately, and keep up their spirits and courage, retain their health much longer, in spite of the badness of their food. That scurvy does not depend exclusively upon the use of salt food, and want of fresh provision, is proved still more forcibly, and moreover, the hypothesis regarding the preponderance of the salts of soda over those of potash in scorbutic blood is overthrown by the fact, that it also appears among people whose diet is almost entirely vegetable, but who suffer from destitution, and live in cold, moist cellars, as is the case in northern countries, especially in Russia (land-scurvy).

The repeated outbreaks of scurvy among the inmates of garrisons and prisons, and other humanely and conscientiously kept institutions, is a remarkable fact, and one which cannot always be ascribed to the influences above mentioned.

Anatomical Appearances.—The bodies of persons who have died of scurvy, if the disease has been of long duration, exhibit ex-

treme emaciation and a moderate œdema, particularly of the lower extremities. The integuments are of a dirty, ashy hue, and are generally covered by dry detached epithelium. Here and there extravasations of variable size are found in the tissue of the cutis as well as in the subcutaneous and intermuscular connective tissue. Besides the liquid effusions of blood, hard coagulated infiltrations, stained red by an admixture of blood, are almost always found beneath the skin, between the muscles; more rarely within the muscles themselves. The blood is remarkably liquid and dark in color. The walls of the vessels and the tissues about them are deeply stained by it. In the cavities of the pleura, pericardium, peritonæum, and articular capsules, there is almost always a serous or serofibrinous effusion, with an admixture of blood. The lungs are more or less compressed by the pleural effusion, while the uncompressed portion is the seat of a bloody œdema. The liver, spleen, and kidneys, are studded with ecchymoses, and appear relaxed and filled with blood, which also is extravasated, and infiltrates the tissues. Ecchymoses also exist between the serous and muscular coats of the intestine. The mucous membrane of the intestine is reddened, swollen, and relaxed, and sometimes is in a state of follicular ulceration.

Symptoms and Course.—The first signs of scurvy are usually those of a general cachexia; the patients complain of great debility and lassitude, and particularly of a sense of excessive weight in their lower limbs. Their spirits are deeply depressed, they can no longer devote themselves to the slightest work, and are excessively sad and despondent. At the same time the face of the patient loses its bright hue and grows pale and muddy, the lips acquire a bluish, livid tinge, the eyes sink into their sockets, and are surrounded by a blue ring. These symptoms are usually accompanied by darting or piercing pains in the limbs and joints, which might easily be mistaken for rheumatic pains. It is not usual for the characteristic local manifestations of scurvy to show themselves until the precursory symptoms have continued for some days or even for some weeks; although *Cejka* has often observed the disease to commence locally, the signs of cachexia not appearing until the gums had grown very sore, and all the body was covered with scurvy-spots.

In our first volume we have described the scorbutic sore mouth in detail. It is the most common and generally is the first of the local symptoms of the disease. There are exceptions, however, to this rule also. *Cejka* mentions instances in which sugillations, tense œdema of the feet, and hard, painful infiltration of the connective tissue, preceded the bloody œdematous relaxation of the gums. The effusions of blood into the tissue of the skin, mentioned above, sometimes take

the form of small round petechiæ, sometimes of vibices, and sometimes of extensive ecchymoses. They almost always appear first upon the lower extremities, and afterward spread over the rest of the body. Regions exposed to the action of trifling mechanical violence are especially liable to become the seat of the ecchymoses. In the epidemic in the house of correction at Prague, described by *Cejka*, the influence of mechanical action was strikingly apparent. In most patients the hollows of the knees were most affected, but, in wood-cutters and in persons who worked at the spinning-wheel, the right arms were attacked. Wool-combers and laundresses suffered in their forearms; women in the place where their garters pressed. The spots which at first were violet, and of almost a blackish brown, pass through the well-known changes of color peculiar to extravasations, blue, green, yellow, etc. When new spots appear, while the old ones are fading, dark-blue and greenish-yellow spots are found simultaneously upon the skin. Sometimes circumscribed effusions beneath the epidermis give rise to blebs filled with a bloody liquid (purpura bullosa, pemphigus scorbuticus), which, if they burst and are treated carelessly, may result in obstinate ulcers. Ulcers also form in some patients after the receipt of trifling injuries. They are characterized by the flabby, spongy, bleeding granulations which cover their surface. The hard infiltration of the subcutaneous and intermuscular areolar tissue forms rounded tumors beneath the skin, varying in size from that of a hazel-nut to that of a fist, situated usually upon the lower, more rarely upon the upper extremities, and upon the belly, throat, and cheeks; they cause more or less pain, and are covered by a skin which either retains its normal color or else is suffused by blood. Sometimes the indurations are extensive and diffuse, covering the hams, the calves of the legs, and the inner surface of the thighs. They are hard as a board, and, owing to the pressure which they exert upon the muscles, they render all movement of the affected limb impossible. The cutis remains immovable above this diffuse induration, and is either of normal appearance or is suffused with blood. Besides the bleeding from the gums, which, though not always very profuse, is seldom absent, hæmorrhage from other mucous membranes occurs in some patients, especially epistaxis, hæmoptysis, metrorrhagia, and bleeding from the bowels. Ecchymosis of the conjunctiva and effusions into the chambers of the eye, with malignant ophthalmia, have occasionally been noticed.

The inflammations of internal viscera, particularly scorbutic pericarditis and pleuritis, do not differ in any characteristic or peculiar manner from primary inflammation. Voluminous effusions are often rapidly deposited, putting the patient in danger of suffocation; but I have also witnessed the rapid and unexpected reabsorption of large collec-

tions in the pleura and pericardium of scorbutic persons. Owing to the variety and irregularity in the succession of the symptoms of this disease, the few cases which have come under my observation have presented very different aspects.

Its course is chronic, and, if the pernicious influences under which it has arisen be not allayed, it is often extremely tedious. In these protracted cases the prostration of the patient becomes excessive; sometimes he faints with every attempt to sit upright; he complains of a distressing palpitation and great dyspnœa; the action of the heart grows very weak and rapid, extensive œdema arises in the subcutaneous connective tissue, while the soreness of the gums, the ecchymoses, the brawny infiltration of the areolar tissue, and the other local affections, increase greatly in extent and intensity. A marked feature of the disease consists in the rapidity of its abatement and the sudden transformation from a condition of the utmost distress to one of relative convalescence, often terminating in full recovery, when the patient is rescued from noxious influences, and placed in a condition favorable to a cure. It is true that, under such circumstances, their recovery is very slow, and they always retain a great tendency to relapse.

A fatal termination of scurvy occurs either late in the disease from extreme exhaustion and general dropsy, or else it takes place earlier and before the prostration has acquired much intensity from pleurisy, pericarditis, profuse bloody diarrhœa, or other complications.

Treatment.—In modern days the sea-scurvy has become rare, owing to the shortening of voyages, and to the better supplying of ships, especially by the provision now made of lemon-juice, sauer-kraut, and hermetically sealed vegetables. Land-scurvy also, which formerly was of much more common occurrence, has become a rare disease in this country, from the wholesomeness of the dwellings and the improved diet which paupers now enjoy, thanks to the progress of modern culture, so that a prophylaxis against scurvy is no longer talked of. Indeed, precautionary remedies are now superfluous, excepting when several cases of scurvy break out in a barrack, or workhouse, or similar institution, causing apprehension of an epidemic appearance of the disease. These precautions consist in the most scrupulous attention to cleanliness, warmth of clothing, ventilation of apartments, exercise in the open air, a sufficient allowance and proper choice of food, which should consist of fresh meat, and, if possible, also of fresh vegetables, and salad. Good beer should also be provided, and, where this is unobtainable, brandy-and-water should be administered. The cost of such attentions is amply compensated for, if we succeed in putting a check upon the disease.

In cases of pronounced scurvy, the freshly-expressed juice of certain

plants, especially that of the order of crucifera, such as water-cresses, cabbage, mustard, radishes, horse-radishes, spoon-wort (scurvy-grass), and the like, is extremely beneficial, while the extracts of the same plants are quite useless. The healing effect of a fresh vegetable diet upon scurvy is much more positively ascertained than is the dependence of the disease upon a want of such nourishment. Next in virtue is the juice of certain acid fruits, especially that of lemons and oranges, although that of cherries, currants, apples, and the like, is also serviceable. The mineral acids are of no benefit whatever. The bitters and aromatics, also, are of a questionable and certainly of a very secondary value. The barm of beer, also, has a great reputation as an anti-scorbutic, when given to the amount of six or eight ounces daily. It is very important that the rules as to diet and regimen, laid down above, in treating of the prophylaxis, should be carefully observed. The local treatment of the sore mouth has been discussed already. For the ecchymoses and infiltrations, lotions and compresses wet with aromatic vinegar, spirits of camphor, and the like, may be applied. The hæmorrhages and internal inflammations are to be treated upon general principles, due regard always being had to the prostrate condition of the patient.

CHAPTER III.

PURPURA HÆMORRHAGICA—THE "MORBUS MACULOSUS" OF WERLHOF.

Etiology.—The symptoms of the spotted disease of *Werlhof* resemble those of scurvy, as far as regards the appearance of extravasations of blood from the capillaries of the skin and mucous membrane; but the hæmorrhages are not accompanied by any affection of the mouth, nor by the peculiar subcutaneous and intermuscular infiltrations, nor the hæmorrhagic inflammations of the serous membranes, which we know to be symptoms of scurvy.

The reason for the tenderness of the capillaries in *Werlhof's* disease is obscure. The extension of the hæmorrhages over various parts of the body, its frequent occurrence among feeble and debilitated persons, and among convalescents from severe illness, and its appearance in those who dwell in damp, unwholesome lodgings, or in other unfavorable conditions, make it probable that the disease of the blood-vessels depends upon an improper state of this nutrition, or upon insufficient or bad nutriment. On the other hand, robust, powerful individuals, living under the most advantageous circumstances, and

who do not offer the slightest reason for a defective state of blood, are also sometimes attacked.

Symptoms and Course.—Sometimes the first symptoms of *Werlhof's* disease consist in the appearance of numerous purpuric spots upon the skin; in other instances the hæmorrhages are preceded for some days or weeks by digestive derangement, languor, debility, but never by the signs of severe cachexia, such as generally precede the onset of scurvy. The purpuric spots, though small, are generally numerous. Their most common seat is upon the extremities and body, although the face is often speckled by numerous petechiæ. While the first set of petechiæ are passing through the blue and green stages, new blood-red ones make their appearance. Now and then hæmorrhages from the surface are observed; but such phenomena cannot properly be called bloody sweat (p. 453). Minute, punctiform ecchymoses are also found upon the mucous membranes, especially those of the mouth and fauces. Epistaxis, hæmatemesis, hæmaturia, and bloody evacuations from the bowels, are of far more common occurrence in this disease than in scorbutus. When it attacks an otherwise healthy and vigorous individual, and when the hæmorrhages do not recur too frequently, the undisturbed condition of the patient's general health often forms a marked contrast with the objective symptoms. Very numerous and oft-repeated hæmorrhages may result in intense anæmia, a tendency to syncope, dropsy, and even death. In previously healthy persons, however, such a termination of the disease is rare. As a general rule, the affection ends in recovery after a duration of between two and four weeks.

Treatment.—The customary treatment of *Werlhof's* disease, which originated with *Werlhof* himself, consists in the administration of sulphuric acid and quinine. Although there is no proof that the use of these articles exerts any decidedly beneficial influence upon the course of the disease, yet, not to be too skeptical, and for want of more certain remedies, it is advisable to give the dilute sulphuric acid or the elixir of vitriol in doses of ten or twelve drops every two hours in the beginning of the disorder, followed at a later period by a decoction of bark with an addition of sulphuric acid. In cases of profuse epistaxis which do not readily subside under the application of cold, the tampon must be employed promptly, since the longer the bleeding is allowed to last, so much the more obstinate does it become. For the hæmatemesis, bits of ice, alum-curds, and cold compresses to the abdomen, should be employed; for hæmaturia, large doses of tannin. In extreme anæmia it is of importance that the patient should preserve a horizontal attitude as a precaution against swooning.

CHAPTER IV.

PROGRESSIVE PERNICIOUS ANÆMIA.

The old names of "essential," "idiopathic," "fatal anæmia," etc., probably referred to the disease now called "progressive pernicious anæmia." Little is known of the causes of this gradually progressing disease, which is nearly always fatal. It is most frequent in middle age, but occurs later, as well as in childhood. It is more frequent in females, especially after confinement or during pregnancy, in which case it is apt to induce miscarriage, followed quickly by death. Sometimes the disease can be ascribed to no cause; at others it may be referred to bad nourishment, repeated losses of blood, diarrhœa, etc. The onset is usually insidious, with great paleness of the skin and mucous membranes; if there is no fever, the nutritive condition of the patient may be little disturbed. The patients are very feeble, with great tendency to faintness, dyspnœa, palpitation, blood-murmurs, and gastric disturbances, vomiting being very common. The spleen and lymphatic glands are not swollen, wherein it differs from leuchæmia, nor does the skin have the brownish color of Addison's disease. There is no regular fever curve; indeed, there is no fever till the disease is far advanced; autopsy has revealed no sufficient cause for the fever.

Often, especially late in the disease, there may be epistaxis, petechiæ, ecchymoses on the legs, and hæmorrhages from the female genitals, from the stomach, air-passages, etc., or into the tissue of the heart, the pericardium, dura mater, brain, retina, etc.; the latter are said to be quite frequently discoverable on ophthalmoscopic examination of the region of the papilla as reddish spots or radiating striæ; it is said that these spots are characterized by peculiar gray or white specks. These hæmorrhages are not, however, limited to this disease; they may occur in various other forms of anæmia; they may be due to the retinal vessels being diseased and being ruptured by vomiting.

As pernicious anæmia begins very gradually, and is not at first detected, its duration cannot be determined; it may prove fatal within a few weeks from the time of its discovery, but usually runs on for months or even years. All cases do not prove fatal.

The diagnosis is difficult in the commencement. It is said that even in the early stages some of the blood-corpuscles are very small, but this is a disputed point. The anæmic symptoms cause it to resemble leuchæmia, but in the latter there is marked increase of the

white corpuscles, and in pseudo-leuchæmia there is swelling of the spleen and lymphatic glands. Cases occur where it is impossible to make an accurate diagnosis, especially when an enlargement of the spleen accompanies the pernicious anæmia. There seem to be cases of transition between this and other diseases. *Litten* says that changes of the medulla of the bones are found in pernicious anæmia, just as in medullary leuchæmia; and time must teach us how far this is a constant process, and whether the real lesion in this affection is a disease of the medulla of the bones, as that of the spleen and lymphatic glands is in pseudo-leuchæmia.

Cases where cachectic states accompany undiscovered cancers may be mistaken for pernicious anæmia.

Treatment has proved unsatisfactory; even the cases ending in recovery seem to have done so spontaneously. We can do little more than remove injurious influences, and give good diet, air, and tonics, especially iron.

It has been claimed that great benefit has followed the administration of phosphorus, but other observers deny the efficacy of this remedy. In some of these forms of anæmia the employment of arsenic by the mouth and subcutaneously is reputed to be very efficacious, but it has not yet been sufficiently tried to enable us to say in which cases it will do good and in which it will fail. It seems quite certain that some patients who are not benefited by administration of iron, will improve if the iron is continued while inhalations of oxygen gas are given in doses of two or three gallons diluted with air several times daily.]

CHAPTER V.

HÆMOPHILIA (HÆMORRHAGIC DIATHESIS).

ETIOLOGY.—The names hæmophilia, hæmorrhaphilia, are applied to a congenital hæmorrhagic diathesis distinguished either by the unusual obstinacy of traumatic hæmorrhage or a tendency to spontaneous bleedings. Hitherto no abnormity capable of accounting for the symptoms has been detected either in the blood or the vascular walls of the patient, although in a small number of cases the walls of the blood-vessels have been found to be remarkably thin and delicate. In most cases of hæmophilia the disease is hereditary, that is, the patient descends from a family one or more of whose members in preceding generations have suffered from the same affection. There are instances in which it has been transmitted through four generations; others in which a generation has been "skipped," the grandchildren having hæmophilia, but not the children. It is rare for every member of a family to inherit this dangerous disorder, and the daughters seem to remain free from it with especial frequence. There also are well-authenticated observations which leave no doubt of the occurrence of congenital hæmophilia not dependent upon hereditary predisposition.

SYMPTOMS AND COURSE.—Until the discovery is made that a trifling wound will give rise to an irrepressible and dangerous loss of blood, there is no symptom to warn the patient of his perilous condition. Some observers, indeed, claim that this class of patients are distinguishable by the remarkable delicacy of their complexion, their superficially situated and conspicuous veins, blonde hair, and blue eyes, and, in one family that I know of, this description is equally applicable to the whole of them, including the female members, who are exempt from the disorder. Other observers, again, state expressly that the patient's appearance presents no perceptible characteristics.

The accidents which give rise to these alarming hæmorrhages are generally extraction of a tooth, a puncture, a small cut or laceration, and it would almost seem as if such injuries were more dangerous than severe wounds. The blood oozes out as if from a sponge, although no bleeding vessel is discoverable; all attempts to stanch the bleeding are in vain, and it persists for days. The blood, which at first is normal, gradually grows thin and watery, forming small, loose coagula. At last the complexion of the patient acquires a waxy pallor, the lips lose their color; syncope and other signs of exsanguinity occur, and the patient may perish in a few days. More commonly, however, the bleeding ceases, and the patient, in a state of utter exhaustion, slowly recovers from the effects of his enormous loss of blood,

which often amounts to many pounds. Besides these dangerous external hæmorrhages, extensive extravasations occur beneath the skin, in consequence of the slightest contusion. *Wunderlich* tells of a boy, who, after receiving a flogging at school, came home black and blue, and so covered with stripes and welts that a charge was preferred of cruelty. It afterward appeared that he had the hæmorrhagic diathesis.

Spontaneous bleeding or hæmorrhages without assignable cause do not generally take place until after the patient has suffered repeatedly from traumatic hæmorrhages. They generally proceed from the nose; but likewise arise from the bronchi, stomach, intestines, and kidney, and may also occur in the substance of the skin and subcutaneous areolar tissue. They are generally preceded by molimina, such as palpitation of the heart, stupor, signs of cerebral congestion, pain in the limbs, and, in some cases, painful tumefaction of the joints, particularly those of the knee and ankle. There have been instances in which the bleeding from the navel-string could not be stanched, but more usually it is not until the period of dentition, and sometimes after the sixth or eighth year of life, or later, that the diathesis betrays itself by a dangerous hæmorrhage. Most patients die young—few surviving the period of childhood. Cases are known, however, in which the patient has lived to a good old age, the tendency to bleed diminishing, or ceasing altogether, as life advanced.

Treatment.—No remedy is known likely to prove efficacious in congenital hæmorrhagic diathesis, and we must therefore confine our efforts to a careful regulation of the habits, and the removal of all noxious agencies; so that, perhaps, by improving the general condition of the constitution, this dangerous diathesis may subside. Of course, all wounds must be scrupulously avoided. When the bleeding occurs in spite of such precautions, besides the ordinary hæmostatic articles, among which steady pressure and the actual cautery are the best, glauber salts in cathartic doses should be prescribed, and, when the bleeding threatens life, two to five grains of secale cornutum every half-hour. These two prescriptions have been of great benefit in some instances.

CHAPTER VI.

SCROFULA.

Etiology.—The term scrofula signifies a morbid (cachectic) condition of the system, manifested by a remarkable liability to certain forms of nutritive disorder of the skin, mucous membranes, joints, bones, organs of special sense, and, above all, the lymphatic glands.

A person having merely a tendency to such diseases may also be called scrofulous, although not actually suffering from any one of the above symptoms.

The hypothesis that scrofula depends upon a faulty composition of the blood (dyscrasia), and that the lesions found in scrofulous persons were due to the deposit in the tissues of a matter circulated by the blood, and called a "scrofulous material," is almost universally abandoned. The alterations which take place in the skin, mucous membrane, joints, bones, and organs of special sense, are of an inflammatory nature, and cannot be distinguished from similar affections of a non-scrofulous character, excepting by their intractability and the tediousness of their course. It is impossible to point out any characteristic features in a scrofulous eruption or arthritis, or to find any difference between them and similar non-scrofulous affections. Even the caseous metamorphosis, to which the inflammatory products are so prone, is by no means pathognomonic of scrofula, but is common to all diseases of a chronic character which have a tendency to disintegration or destruction. Notwithstanding, however, that such inflammation presents no distinguishing mark whereby its scrofulous nature may be recognized, yet there will rarely be any doubt as to whether or not a case of this kind is entitled to the name. If its exciting cause have been so slight as to be overlooked; if we are informed that "the disease came on of itself;" if this attack, or other similar ones, have arisen repeatedly without assignable cause; if it be accompanied by a series of other disorders, especially chronic inflammation and persistent enlargement of lymphatic glands, it is to be called scrofulous. If, on the contrary, there be known external conditions, whose action upon the system sufficiently accounts for the occurrence and the obstinacy of the affection, without necessitating the supposition that there is any special morbid tendency; if it exist independently, and be uncomplicated with chronic enlargement of the lymphatics, then it is not to be called scrofulous, notwithstanding the great similarity, nay, the absolute resemblance, of its external symptoms to those of a scrofulous disorder.

In its origin, scrofula, perhaps, is quite as often a congenital malady as an acquired one after birth.

Congenital scrofula is particularly common among offspring of scrofulous parents. There are families, nearly or quite all the children of which inherit the disease. This hereditary form of scrofula is closely allied to that in which parents were tuberculous at the time of begetting the child, or in which the mother was so during pregnancy, or else suffered from cancer, tertiary syphilis, or some other malady, as well as that form of scrofula occurring in children begotten of aged

parents. As we know that many of the bodily and mental traits of parents are transmitted to their progeny, it will not seem extraordinary that children of feeble, sickly fathers or mothers should have a greater tendency to disease than those whose progenitors are vigorous and healthy; but we are totally unable to account for the fact that scrofula is also very prevalent in the children of parents who are too closely related to one another by blood. It must be added, moreover, that it is not every child of scrofulous, sickly, feeble, or superannuated parentage, and not all of the issue of the marriages of near relatives, who suffer from congenital scrofula. Indeed, many children thus born are healthy, and without any decided tendency to disease; and, on the other hand, this malady often attacks the offspring of parents entirely exempt from the action of any of the above predisposing agencies.

Acquired scrofula generally arises as a result of pernicious influences which have impeded the healthy development of the system during the first years of life. First of all, among these, stands improper nourishment; a coarse diet, containing but little nutriment in comparison with its bulk, being very properly held in especially evil repute. The earlier this injudicious feeding of an infant commences, so much the greater is the danger that it will become scrofulous; hence, the children fed on pap furnish a very important contingent to the army of scrofulous persons. Want of fresh air and exercise exerts an influence as baneful as that of improper food. *Hirsch* has collected a large number of facts, proving that, in foundling hospitals, orphan asylums, boarding-schools, factories, and similar institutions, the continual abode in a badly-ventilated atmosphere, saturated with steam, and poisoned by animal effluvia and the products of putrefaction, is in the highest degree favorable to the development of scrofula; and that, according to the experience of these institutions where there is no lack of cleanliness, good food, and warm clothing, the above pernicious agents alone suffice to induce the disease.

It springs most frequently, however, from the combined effects of all these different anti-hygienic influences. Although we have declared the chief cause of scrofula to be the impairment of the normal development of the system during childhood, through the action of the above-mentioned noxious agencies, yet it must be added that, in prisons, poor-houses, and workhouses, the disorder also breaks out among adults subjected to the simultaneous effects of bad nourishment and want of fresh air. The development or reëstablishment of scrofula is, likewise, a not unfrequent sequel to certain acute and chronic diseases; and this is all the more likely to be the case, the younger and the more undeveloped the subject of disease happens to be. Among

the diseases of childhood, measles is especially mischievous in this way.

Anatomical Appearances.—Having already stated that scrofulous inflammation has no peculiar characteristics, it would now be absurd to attempt to describe the anatomical alterations of the skin and mucous membranes, induced by scrofulous eruptions and catarrh. And, indeed, we may refer altogether to the article upon cutaneous diseases for a description of the anatomical alterations of the skin; and to other parts of this work, for an account of the lesions found in the various mucous membranes, merely adding that in scrofula the inflammatory products are generally richly provided with young cells, which accounts for their tendency to caseous metamorphosis. A description of the esions of the bones, joints, and organs of special sense, particularly those of the eye, observed in scrofulous subjects, and which, likewise, are not marked by any distinctive peculiarity, belongs to the province of surgery and ophthalmology. The latter science has anticipated the others by the discovery of the illusory nature of the oft-described symptoms of scrofulous ophthalmia.

The alterations which take place in the lymphatics of scrofulous subjects require further attention: first, because the disorders of these organs as yet have received but cursory notice, no special section of this work having been devoted to their consideration; secondly, because, in persons thus afflicted, disease in the lymphatics is so common that, among the laity, scrofula is commonly spoken of as having "enlarged kernels," or simply "having kernels."

In the first place, it happens more frequently in scrofulous persons than in non-scrofulous ones, that the lymphatic glands around an inflammation of the skin or mucous membrane become the seat of an inflammatory process, which has extended along the lymphatic vessels from the skin, and has involved the glands. The connective tissue around the gland next participates in the inflammation of the glandular parenchyma, and abscesses and ulceration ensue, which, like all abscesses and ulceration of glandular substance, are very intractable, and often heal, leaving an irregular, ragged, unsightly scar. Owing to the extreme frequence of scrofulous eruptions upon the face and scalp, and of scrofulous otorrhœa, the lymphatic glands of the neck are the most common seat of this form of inflammation and ulceration.

Besides these, however, a chronic non-inflammatory enlargement of the lymphatic glands is so often observed in scrofulous subjects, that their presence may almost be regarded as pathognomonic. They may attain a very considerable size, and, when several of them are clustered together, they form huge, knotted cords, or shapeless lumps. The individual tumors are regular in form, smooth of surface, and of a tol-

erably firm consistence. No foreign elements can be detected in them under the microscope. They are the product of simple hypertrophy, or, rather (since it is the cells that are multiplied, and not the tissue of the stroma), of a "cellular hyperplasia." But, as the number of cellular elements contained in a lymphatic gland, even during health, is a very variable one, the complete subsidence of the enormously enlarged glands of scrofulous subjects, which enlargement is entirely due to an excessive accumulation of cells, will not seem extraordinary. Nevertheless, the gradual diminution of the tumor, with its final return to its normal dimensions, is not the sole termination of this form of glandular affection. Sometimes the tumors, which at first were the result of mere hyperplasia, sooner or later become inflamed. The surrounding areolar tissue takes part in the inflammation, after which, it is no longer practicable to distinguish the individual glands in the general mass. The skin becomes adherent to the tumor, and cannot be made to glide over it. Suppuration and the formation of abscesses generally follow slowly and gradually upon the inflammation, and the skin, having become extremely attenuated, is perforated by the pus, leaving a sinuous ulcer, with undermined edges. In other instances, inflammation and suppuration only take place at a solitary point in the tumor; and the matter formed at this spot does not burst through the capsule of the gland, but thickens, and undergo cesaseous metamorphosis. In such a case, if the swelling subside, the caseous mass projects above the surface of the gland, giving it an irregular shape. A partial or complete caseous degeneration may also take place in the hyperplastic mass, without any previous inflammation or suppuration, the overcrowded cells drying up, and suffering "anæmic necrosis" (necrobiosis). Such an occurrence at circumscribed points likewise renders the contour of the gland irregular and angular. The caseous deposit may subsequently become calcified, but, since it acts as an irritant upon surrounding parts, like any other foreign body, it may subsequently give rise to an obstinate inflammation and suppuration. When an entire gland underwent caseous metamorphosis, the process used formerly to be called infiltrated glandular tuberculosis. If, on the other hand, the deposit of caseous matter was limited, the gland was supposed to be affected by miliary tuberculosis, although there were no miliary tubercles to be found in it. The most frequent seat of scrofulous hyperplasia of the lymphatic glands, like that of scrofulous inflammation, is in the neck, especially behind the ear, and under the lower jaw, extending thence to the shoulder. The bronchial and mesenteric glands are also very often the seat of scrofulous hyperplasia, with all its consequences, as above described. The terms scrofulous catarrh of the bronchi or intestines is perfectly appropriate to bronchial

and intestinal catarrhs, accompanied by enlargement or caseous degeneration of the bronchial or mesenteric glands.

Symptoms and Course.—In many instances the scrofulous cachexia betrays itself by the patient's habit of body, which will often arouse suspicion of his delicacy and of his feeble power of resistance against disease, long before any actual morbid tendency (diathesis) is, as it were, officially announced by the occurrence of frequent and obstinate attacks of illness induced by causes so insignificant as to have escaped observation.

The "scrofulous habit" is marked by a deficience of blood and by a bad nutritive state of the more important and more highly-organized tissues; sometimes accompanied by an accumulation of fat in certain regions, especially in the upper lip and nose. When the imperfect nutrition is accompanied by an over-production of fat, there seems to be an indolent state of the processes of constructive and destructive assimilation; but when not only the skin and the muscles, but also the subcutaneous fat, is in a state of imperfect development, it is probable that these processes are in a condition of unnatural activity. Based upon these differences, in the patient's bodily habit, a classification of scrofula into the torpid and erethitic forms has been made. *Canstatt* very aptly describes the torpid scrofulous habit in the following words: "An unusually large head, coarse features, a thick chin, swollen abdomen, enlarged cervical glands, and flabby, spongy flesh;" and depicts the habit of erethitic scrofula as follows: "A skin of remarkable whiteness, with a tendency to redden easily, and through which the rose-pink or bluish subcutaneous veins are visible, a deep redness of the cheeks and lips, blueness of the thin and transparent sclerotica, which imparts a swimming and languishing look to the eyes. The muscles of such persons are thin and soft, and their weight is light in proportion to their stature, indicating a slightness of their bones. The teeth are handsome, and of a bluish lustre, though long and narrow; the hair is soft." There is no lack of examples of either of these forms of disease, and though it may not be possible immediately to assign every case to one or other category, yet in practice it will be well to adhere to the classification into torpid and erethitic scrofula, since, as we shall find by-and-by, we thereby obtain valuable data for the establishment of therapeutical indications.

Scrofula, although principally a disease of childhood, rarely declares itself in the first year of infancy, excepting through a few faint tokens. At the period of puberty scrofulous diseases usually subside, and with them the scrofulous habit more or less completely disappears. More rarely, exposure of the body to pernicious influences at this time excites the subsiding scrofulous tendency to renewed activity, or even induces a

relapse of disease which has been dormant for years. We have already stated that, under such circumstances, persons, who during childhood did not suffer from the disease, are attacked by it later in life.

It is impossible to describe scrofula briefly, and at the same time comprehensively; for its various manifestations combine in the most diverse manner: in one case, this group of symptoms, in another, that one assuming prominence; and since many patients, notwithstanding the tedious course of their malady, remain free from symptoms which form the most important feature in other cases. And although it is little to be doubted that the localization of scrofulosis (if we may use the term) depends either upon the action of causes which are especially hurtful to the organ attacked, or else upon the morbid susceptibility of the organ to influences whose effect is universally injurious, nevertheless we can seldom tell why it should be marked in one case by a scrofulous exanthema, in another by an ophthalmia; why, in a third, a disease of bone should prevail; and it is equally inexplicable why the inflammation and hyperplasia of the lymphatic glands should be more extensive and obstinate in one case than in another. It has not been determined even whether there be such a thing as primary hyperplasia of the glands, or if this process, like the inflammation, be always of a secondary character, proceeding from irritation transmitted to the gland from some neighboring focus of inflammation. As the reality of the latter fact is susceptible of proof in a great majority of instances, it is not improbable, where it cannot be proved, that the irritation formerly existed at the point of origin of the lymphatic vessels involved, but that it has already subsided; for it is a rule that glandular enlargements long outlast the morbid process which has induced them.

The cutaneous eruptions which are the most common, and often the earliest symptoms of the diathesis, are usually situated upon the face and scalp. They generally belong to the form of dermatitis, in which an exudation, more or less filled with cells, is effused upon the surface of the cutis, and which nowadays are known as eczema and impetigo, and used formerly to be called tinea and porrigo. The more destructive affections of the skin—such as lupus—do not usually appear until a later period of life.

Scrofulous inflammation of the mucous membrane is most apt to appear in the vicinity of the natural orifices of the body, where it readily implicates the neighboring skin, especially if it be moistened by the superabundant secretions. Thus scrofulous coryza is usually complicated with eczema of the upper lip; inflammation of the external auditory passage with eczema about the ear; catarrhal conjunctivitis with eczema of the cheek; conversely, the cutaneous eruptions about an orifice often spread to its mucous membranes, thus inducing

coryza, otorrhœa, conjunctivitis, or ulceration of the cornea. Catarrhs of the intestine and bronchi, and the rarer affections of the genito-urinary apparatus by their obstinacy, may readily excite a suspicion of a more malignant destructive process; and indeed scrofulous catarrh of the bronchi, by extending into the air-cells of the lung, and by inducing caseous metamorphosis, with subsequent destruction of the pneumonic deposits, not unfrequently terminates in pulmonary consumption. Besides, it often happens that enlarged bronchial glands soften and break down, forming vomicæ, which discharge their contents into the bronchi. Scrofulous ulceration of the bowels has already been described in detail.

Scrofulous inflammation of a joint sometimes takes the form of a simple hydrarthrosis, sometimes that of a so-called tumor albus, while at others it assumes the nature of a malignant arthrocace, accompanied by suppuration, caries of the articular surfaces, burrowing of pus, and the establishment of fistulæ. The disease of the bones sometimes begins in the periosteum, sometimes in the bone itself, now presenting the character of periostitis and ostitis, and now that of caries, necrosis, or of the two combined.

As long as the existence of cheesy masses was regarded as characteristic of the tuberculous nature of a disease, it was of course necessary to ascribe many of the inflammations of the joints and bones of scrofulous persons to a complication of scrofulosis with tuberculosis.

Among the organs of special sense, the eyes and more particularly their superficial tissues, the Meibomian glands, the conjunctiva palpebrarum, the conjunctiva bulbi, and the cornea, are often afflicted by obstinate inflammation. Indeed, in doubtful cases, the presence of opacity or of scars upon the cornea, has been accepted as important evidence that a patient has been scrofulous during childhood. In the nose, excepting the rare instances in which that organ is destroyed by lupus, scrofulous inflammation merely takes the form of an obstinate coryza, which, however, is hardly ever absent. In the ears, besides the inflammation of the external auditory canal, already alluded to, caries of the petrous bone, with all its mischievous consequences, is apt to ensue.

The objective symptoms of scrofulous disease of the lymphatic glands have been described above. The adenitis and the phlegmonous inflammation of the parts about them may be attended with great pain and by more or less of fever, with evening exacerbations. In consequence of this, not only does the patient's general health suffer, but, if the inflammation and its attendant pain continue for weeks and months, as they often do, his strength is consumed, and his nutritive condition is greatly impaired. After the abscesses which form have

burst spontaneously, or have been lanced, and if no new centre of inflammation have formed meantime, the fever gradually abates, the patient's strength returns, the emaciation ceases, often long before the ulcers have begun to heal, and while they still secrete large quantities of pus.

Glandular tumors depending upon cellular hyperplasia are not attended either by pain or fever; but, when very voluminous, particularly when seated upon the neck, they not only produce great deformity but hinder the mobility of the head. Hitherto it has not been ascertained whether scrofulous enlargement of the lymphatic glands exerts any detrimental influence upon the composition of the blood, whether the cellular elements destined for the blood are retained in the gland, or whether scrofulous glands affect the quality of the blood as the glands of leucæmia do, but in a far slighter degree.

The progress of scrofula is tedious and treacherous, and nearly always is marked by periodical alternations of improvement and aggravation. Either the same series of symptoms recurs again and again with renewed severity, or else, the former set having abated, or subsided, new ones arise. Complete recovery from scrofula is a very common occurrence, especially if we include the cases in which no trace of the disease remains excepting a peripheral speck upon the cornea, an unsightly cicatrix, or other mark, which does not disturb the general health and comfort. Death rarely results from the lesions to which the epithet of scrofulous is usually given. Of these, tedious disease of the bones and joints, and suppuration of the bronchial glands, are the only ones which endanger life. On the other hand, a large number of scrofulous children die of croup, hydrocephalus, and other acute maladies, for which such subjects have a predisposition quite as decided as for the affections to which the term scrofulous usually is applied. Finally, it should be mentioned that lardaceous disease of the liver (*Budd's* scrofulous tumor of the liver), spleen, and kidneys, often develops during scrofulous disorders, particularly in tedious cases of suppuration and disease of the bones.

Regarding the relationship of scrofula to tubercle, we have already stated that no doubt many scrofulous children afterward become tuberculous; but also that a large number of them escape tuberculosis, especially those in whom the constitutional delicacy, upon which the former affection depends, subsides in after-life, and in whom no caseous deposits remain as residua of scrofulous disease.

Treatment.—The prophylaxis against congenital scrofula lies almost beyond the sphere of the physician. It is devoutly to be desired that scrofulous, tuberculous, sickly, and superannuated persons should not marry at all, and that healthy and vigorous individuals

should not wed their near relatives. But the representations of the physician, that the issue of such alliances are likely to be scrofulous, will rarely deter a man from marrying. On the other hand, it is not only an imperative, but a grateful task to the physician, to endeavor to provide, by proper precautionary measures, against the extension of acquired scrofula. How to fulfil this duty becomes apparent from what has already been said regarding the causes of acquired scrofula. Above all, it should be borne in mind that the development of this disease is promoted, not by one particular noxious influence merely, but by every condition incompatible with health, to which the system, especially during childhood, can be exposed. It often happens that, from the moment when the first tokens of scrofula appear, or even as soon as the parents begin to dread its attacks, the child is carefully deprived of every particle of bread and butter, potato, and the like, and dosed with huge prophylactic spoonfuls of cod-liver oil, while at the same time he is suffered to sit all day long in a close chamber, or upon the benches of an overcrowded school-room. A sufficiency of fresh air and muscular exercise are prophylactic measures of quite as much importance as regulation of the diet is; and we shall here call attention to an error upon this subject, to which we have referred once before while discussing the prophylactic treatment of consumption: namely, the belief that the use of bread and potatoes favors the development of scrofula and tubercle, and the consequent complete privation of children of this kind of food; while in reality the mischief is due to an insufficient supply of animal food—a diet of potatoes and other articles, containing little nourishment in proportion to their bulk, not being injurious unless it forms their sole or at least their principal subsistence. This error is so prevalent, that the discovery that the child has surreptitiously eaten a potato has cost many an anxious mother a sleepless night, and many a child has uselessly been sent to bed hungry on this account.

The treatment of pronounced scrofula, where the prophylaxis has been neglected or unsuccessful, requires, first of all, a careful regulation of the regimen upon principles already laid down. Children with congenital scrofulosis must not be the subjects of experiment with artificial food; but, if the mother have not milk enough, or if she be sickly or feeble, so that, both in her own interest and in that of the babe, she must be forbidden to suckle it, a good wet-nurse must be selected with the utmost care. There is no substitute for the mother's or nurse's milk, and the first year of infancy is perhaps the most important to the constitution of the whole life. When the child is older, the directions as to the diet and the mode of life must be given with the utmost precision. It is not advisable to order that "the patient shall eat but little bread, and a good deal of meat, soup, milk, and the like;" and

that "he must not work too long," and should take "plenty of out-of-door exercise." To be successful in our object, the quantities of food and drink must be prescribed definitely, as well as the number of hours allotted to each form of occupation.

Cod-liver oil has a special and well-merited reputation as a remedy against scrofula, and there are plenty of instances where it has done good service. On the other hand, perhaps no remedy has ever been so much abused as this one. Whosoever supposes that the mere presence of a thick nose, a sore upper-lip, or a bunch of enlarged cervical glands, affords sufficient ground for the prescription of this medicine, will often fail to benefit his patient, and sometimes will do him harm. Daily experience teaches, however, that such is the general belief, and that he who seeks to combat it does not merely fight a windmill. Let any one ask a patient whose scrofula has outlasted his childhood, and who has passed again and again from one practitioner to another, how often he has had cod-liver oil prescribed for him since the time of its first failure during childhood; how many months or years he has taken it; and how much the whole aggregate quantity would amount to; and he will be surprised at the answer. Nevertheless, in all probability, the next physician whom the patient consults will prescribe it again. A most serviceable means of distinction, between the cases in which cod-liver oil is indicated and those in which nothing is to be expected from it, is afforded by the symptoms of the torpid and erethitic forms of scrofula. When the patient's slender frame, the lack of fat beneath his skin, and his accelerated pulse, warrant the belief that his nervous system is in a state of over-activity, cod-liver oil is generally of the most signal benefit. Under its use the plumpness of the body increases, while the general susceptibility of the system, and the diseases consequent upon it subside. These are the cases to which this article owes its name as an anti-scrofulous remedy. But if the patient be clumsy and thick-set; if the nose and upper-lip be enlarged, and the adipose layer over the rest of the body strongly developed; if the action of the heart be retarded rather than accelerated; if the irritability of the nervous system seem unusually obtuse; in short, should there be reason to suppose that the waste of the system is diminished rather than increased, we cannot hope to relieve the disease by means of the oil. Nevertheless, it is precisely this class of patients who in vain have taken such enormous quantities of it in the course of their lives. Besides the oil, and as a corroborant of its effects, so to speak, articles containing a little tannin, such as parched acorns, "acorn-coffee," and home-made infusions of walnut-leaves, are very often prescribed. Such a practice is greatly to be commended whenever there is a chronic catarrh of the intestines embarrassing the digestion and the absorption

of chyle, and where apprehensions are entertained that the oil may aggravate the intestinal disorder. When used in appropriate cases, we prize the efficacy of cod-liver oil, with acorn-coffee, and walnut-tea, most highly, although we strongly deprecate its indiscriminate employment in every form of scrofula; but, as it is requisite that its exhibition should be long continued in order to produce favorable results, certain rules for its administration must be laid down. The disgust for the oil, which in adults is sometimes invincible, is soon overcome by children, who generally speedily cease to fight against the customary dose of two teaspoonfuls daily, and even ask for it themselves when the period for its administration has expired. But, if the treatment be kept up for months without occasional intermission, an unconquerable, and then too generally a permanent aversion to the disgusting medicine will arise, even among children, so that vomiting and retching ensue after every attempt to force it down. This awkward occurrence, which often renders further treatment futile, may nearly always be avoided by interrupting the "cure" for a week or a fortnight after continuing it for four or six weeks. In order to make children take the acorn-coffee as willingly as real coffee, it is sufficient to add a few coffee-beans to the acorns before roasting them.

It is far more difficult to furnish definite instructions for the use of the brine-baths, whose anti-scrofulous virtues enjoy a reputation almost as great as that of cod-liver oil. We know too little about the action of these baths, and about the effect which they produce upon nutrition, and the advantages derived from the salt, iodine, and bromine, which they contain, to enable us to determine upon theoretical principles where they are indicated and where they are unlikely to do good. A calm analysis of the positive and negative effects of brine-baths in scrofula, which would be the best means of obtaining fixed indications for their use, has not, as yet, been made; and the doctors at the baths, who certainly ought to be the best judges of the extent and limits of their healing powers, rarely send away a scrofulous patient as unfit to use the brine. Hence, there is no resource but to send persons, who have in vain tried cod-liver oil and other anti-scrofulous remedies, to Kreutznach, Ischl, Kosen, or Wittekind, or some similar watering-place, in the hope that they may be among those to whom the baths will exhibit their anti-scrofulous virtues, which are by no means illusory; and, if the circumstances of the patients do not admit of this, they must use artificial brine-baths at home.

In recent times the cold-water cure has earned for itself a most favorable reputation as a remedy for scrofula; and, indeed, a series of cases is on record in which complete and perfect cures have been obtained by these means, after all other modes of treatment had been

applied in vain. We certainly are justified in asserting that cod-liver oil treatment cannot be substituted for the water-cure, nor *vice versa;* but we may go still further, and declare that the oil is not only useless in a case adapted for the water-cure treatment, but is absolutely pernicious; and the same holds good regarding the hydropathic treatment in a case where large doses of cod-liver oil are indicated. When we consider that formerly the "decoctions of woods," laxative tisanes, antimonials, mercurials, and other drugs intended to stimulate the emunctories into activity, were much employed in scrofula, and when, after weeks of such treatment, a profuse flow of urine or of sweat was finally induced, or, when the patient began to purge copiously, very good results were sometimes obtained, it will be plain that this treatment (the effect of which is to cause an intensely active destructive assimilation and consumption of the tissues) can only benefit the class of patients in whom these processes are in a state of indolence and inactivity. The hydropathic treatment has a similar though far less pernicious influence, and is of signal benefit in the torpid form of scrofula, that is, in that form where cod-liver oil is powerless, as I have repeatedly had opportunity of satisfying myself.

Iodine and the mercurials are also generally regarded as good antiscrofulous medicaments. It is impossible to hold such views while believing scrofula to consist in a cachexia, marked by a peculiar proneness to disease, and evinced by a series of nutritive disturbances; for one cannot well suppose that the use of iodine or mercury can augment the power of the system to resist noxious influences. I therefore consider it improper to prescribe these articles solely on account of the scrofula, and without fixed indications arising from the peculiarities of the case. At the same time I will not deny that the cases are pretty numerous in which the preparations of iodine and of mercury are indicated. For further details, I refer to the books of local pathology and therapeutics, surgery and ophthalmology, merely alluding to the great benefit derivable from the internal and external exhibition of iodine in the chronic hyperplasia of the lymphatics. In particular, where chronic indolent enlargements of the lymphatic glands form the sole remaining vestige of a former disease, astonishing advantage is often gained by the use of the Adelheid springs and the Krankenheil waters.[2]

CHAPTER VII.

DIABETES MELLITUS—MELLITURIA.

Etiology.—The pathogeny of diabetes still remains obscure. The discovery of the physiologists, that sugar appears in the urine of animals after puncture of the floor of their fourth cerebral ventricle, has not as

yet thrown light upon the mystery. We know that the presence of sugar in the urine, whence diabetes mellitus derives its name, does not depend upon functional abnormity of the kidneys; that the sugar is not formed in them, and that it is excreted from the blood; but we are altogether ignorant wherein the constitutional anomaly consists, in consequence of which a diabetic patient's blood contains sugar, and a healthy person's none. As the various hypotheses offered to account for diabetes are of little practical value, we shall mention a few merely of those most generally entertained.

In the first place, diabetes has been ascribed to an insufficient degree of transformation of the sugar in the blood. Indeed, if the sugar into which the amylaceous substances that have been eaten are converted, and that which is formed in the liver, were to circulate in the blood without undergoing further alteration, and if this substance did not disappear again from the blood during its passage through the lungs, it would necessarily form one of the normal constituents of the urine; hence it cannot be denied that the pathological presence of sugar in the urine may possibly depend in some cases upon a failure of the conditions under which the normal transformation of the sugar takes place. However, as long as we are ignorant as to what these conditions are, we obtain but little aid from the hypothesis that their absence is the cause of diabetes. The assertion, that the non-assimilation of sugar in the circulation of diabetic persons is due to a want of alkalies in the blood, has been disproved. The theory of the existence in the blood of healthy persons of a certain unknown ferment, which induces the assimilation of sugar, but which is absent in the blood of diabetic persons, is untenable.

Others have sought to trace the origin of diabetes to an abnormally accelerated conversion of amylaceous matter into sugar, this being due to a diseased condition of the digestive juices. To this theory there are still greater objections than to the preceding ones. It seems that even in healthy persons the amylaceous ingesta are all changed into sugar (*Trommer* has proved this to be the case even in geese that have been "crammed"), and yet we know that healthy urine never contains sugar even after the ingestion into the system of very large quantities of amylaceous matter, or of pure sugar. But the result of the treatment based upon these hypotheses has afforded the best evidence of their unsoundness. Were the source of diabetes an over-active and immoderate conversion of amylum into sugar, the disease should cease upon the stoppage of the supply of amylum. Experience, however, teaches us the contrary; as, even where for weeks and months the food of the patient has been exclusively animal food, the sugar rarely disappears from the urine. Finally, since *Claude*

Bernard has shown that the liver produces glycogenous substances and sugar, the hypothesis that diabetes depends upon a disease of the liver has gained ground, although the theory is not supported either by pathological or clinical observation. *Claude Bernard* himself thinks that the diabetes induced experimentally in animals, by the "*diabetic puncture*," depends upon the nervous derangement of the liver to which the operation gives rise. As a consequence of abnormal innervation, not only is the production of glycogenous matter augmented, but its conversion into sugar is also hastened. He believes, too, that in the human subject, diabetes mellitus likewise is due to an over-activity of the nerves which stimulate the function of the liver, and considers it probable that, "if it were in our power to galvanize the sympathetic nerve," this would be the best possible mode of treating diabetes symptomatically; the function of this nerve being weakened by the undue activity of its antagonists. The possibility cannot be denied that more glycogenous matter is formed in the liver of a diabetic person than in that of a healthy one; but it is neither proved nor even probable that this anomaly constitutes the only or even the essential lesion of the disease. It is not to be supposed that the pound of sugar which some patients discharge in the course of every twenty-four hours is only a small portion of the sugar which they produce daily, and that a larger portion undergoes that transformation in the blood which, normally as we know, is undergone by very large quantities of sugar, while a smaller portion of it remains, as it were, superfluous, and passes away in the urine. In conclusion, I will present another very specious hypothesis, which really accounts better than any other for the origin of diabetes. According to *Tscherinoff*, the liver-sugar is not made from the glycogen, but, on the contrary, the glycogen is formed from the sugar which arrives in the liver. Hence, instead of *glycogen* (sugar-former), it should be called *glycophthinium*, or sugar-consumer. If the liver loses its capacity to convert the sugar into glycophthinium, the sugar remains in the blood, and thus causes diabetes.

Regarding the etiology of diabetes, our knowledge is equally vague, for although we learn from the history of many cases that the disease has arisen sooner or later after the exposure of the patient to certain noxious influences, yet these influences are so general in their character, and act so often upon the system without being followed by diabetes, that it becomes questionable whether the disease be attributable to their action at all.

Griesinger, after collecting a large number of foreign and domestic cases, has come to the following conclusion: Diabetes occurs much more frequently in males than in females, the proportion being about

three to one. During childhood and old age it is rarer than during the prime of life, the period of greatest frequence being between the ages of thirty and forty in males, and in females between ten and thirty. The disease sometimes appears to depend upon hereditary predisposition. The most commonly recognized exciting causes are: exposure to cold and wet, external violence, concussions of the whole body being a more fertile source of the disease than injuries to the brain and spinal marrow; also immoderate eating of sugar, new wine, and "fruit-must," unfermented juice of fruit, indulgence in immoderate mental exertion, mental depression, intoxication, etc.

Anatomical Appearances.—No constant lesion is found *post-mortem* in the bodies of those who have died of diabetes mellitus. The most common appearances are the following: extreme emaciation, the integument often presenting remains of bed-sores, boils, carbuncles, and sloughs. There is no remarkable lesion in the brain; in the lungs there are almost always tuberculous, or caseous deposits, of variable age, and not unfrequently pneumonia, or gangrene. The heart is flabby and atrophied; the liver usually normal, though sometimes hyperæmic. Hypertrophy of the pancreas occurs with remarkable frequence, considering how seldom this organ is the seat of disease. The walls of the stomach are moderately thickened, through muscular hypertrophy, probably the result of distention. The kidneys are enlarged by hyperæmia, and sometimes are in a state of chronic parenchymatous inflammation (*Bright's* disease).

Symptoms and Course.—The most conspicuous and remarkable symptom of diabetes mellitus consists in the evacuation of enormous quantities of pale urine, whose high specific gravity, contrasted with its limpid aspect and sweetish taste, often alone suffices to place the diagnosis beyond a doubt. It is by no means rare for a diabetic patient, in the course of twenty-four hours, to pass from five to ten thousand cubic centimetres of urine (from five to ten quarts), and, in some instances, the daily discharge has been much larger. No credence should be given to the stories of diabetic patients passing six or eight times this amount, or even more. It is maintained by some that the quantity of urine discharged exceeds the amount of liquid which has been drunk during the meals. If this were true, and unless there were a corresponding waste of the body, we should be compelled to assume that, instead of exhaling liquid through the skin and lungs, the patient actually imbibed moisture in this way from the surrounding atmosphere. Since, as long as the temperature of the body remains higher than that of the air about it, such an occurrence would be incompatible with physical laws, I think it most likely that the observations have been erroneous in the instances where individuals have been reported,

for long periods together, to evacuate a larger weight of urine and fæces than they had taken in of food and drink, without losing in weight. Every investigator and experimenter, who has given his attention to the subject of diabetes, has to suffer from the strong inclination on the part of the patient to deceive him, and to conceal a portion of the liquid that he drinks. In the researches and experiments of *Reich* and *Liebermeister* at the Greifswalder clinic, the results of which have been published by Dr. *Reich*, at first, the quantity of urine and fæces, evacuated by both of the patients under observation, seemed to exceed the amount which they ate and drank. But after they began to watch the patients so closely that they were never left alone for a moment, nor withdrawn from immediate observation, the quantity of ingesta began to exceed that of excreta of urine and fæces.

With regard to the cause of the immense increase in the secretion of urine in diabetes, *Liebermeister* and *Reich* have proved by their experiments that the very large quantity of liquid which they imbibe is not of itself sufficient to account for it. For a number of days, and with great care, they measured out equal portions of food and drink to two patients with diabetes, and a similar portion to a perfectly healthy and trustworthy man (Mr. *Hoffmann*, student of medicine), and measured the flow of urine for every twenty-four hours, from each of them, with the same accuracy. The result showed, that in Candidate *Hoffmann* the secretion of urine was considerably augmented by the very large quantity of liquid which he had swallowed in the cause of science, but that it was far less than that passed in the same time by the sick men. *Hoffmann* passed from five thousand to six thousand cubic centimetres (five to six quarts), while the patients passed from seven thousand to ten thousand. It is possible that the presence of sugar in the blood may increase the power of filtration through the glomeruli of the Malpighian capsules, thereby augmenting the amount of urine secreted. However, this hypothetical explanation of the polyuria of diabetes is superfluous, since it is fully accounted for by *Vogel*, in his classical work upon this disease, as follows: "Whatsoever may be the source of the sugar contained in the blood in diabetes, the necessary consequence of its presence is, that the saccharine serum of the blood greedily attracts to itself, by endosmosis, all the liquids from the parenchyma, and all the water of the food and drink which enters the alimentary canal. The more the blood attracts water, so much the more does it increase in volume, and augment the intravascular pressure. The increase of pressure in the glomeruli of the Malpighian capsules of the kidneys then produces polyuria."

The high specific gravity of diabetic urine, which in mild cases

ranges from 1020 to 1030, and in severe ones from 1030 to 1050, depends almost exclusively upon the quantity of sugar in it; for although the common belief has not proved true, that the absolute amount of urea and salts is reduced in diabetic urine, yet, owing to the enormous quantity of water in which they are dissolved, their relative quantity is very small indeed. *Liebermeister* and *Reich* found that the production of urea of their patients not only was greater than that of healthy persons moderately supplied with mixed food, but that it also exceeded that of a healthy man, who ate as much food as the diabetic patients did. The amount of urea fluctuated in the patients from thirty-two to fifty-five grammes, and in a healthy subject from twenty-nine to thirty-two grammes.

Although the profusion of the urine, its high specific gravity, and its sweetish taste, are decidedly indicative of the presence in it of sugar, yet its positive existence can be easily proved by means of one of the many "sugar-tests." Although it is important to select the most delicate and surest of these, when we wish to determine the presence of very minute quantities of sugar, and although the supposed discoveries of traces of sugar in healthy urine, and in many other pathological and physiological conditions, are in a measure due to our mistaking other substances, having similar reaction, for sugar, yet, when it appears in large quantities, as it does in diabetic urine, *Trommer's* test is quite sufficient to settle the diagnosis. An excess of liquor potassæ is to be mingled with a portion of the urine to be examined; a dilute solution of sulphate of copper is then to be added, drop by drop, as long as the precipitate which first forms will redissolve when stirred. The liquid (which, if sugar be present, will show a blue color) is then to be filtered and warmed. The precipitation of red oxide of copper in the liquid, while still below the boiling-point, furnishes absolute proof that the solution contains sugar. If albumen also be present, it must previously be separated by boiling and filtration. Another more simple test consists in warming the urine after adding a solution of potassa. The presence of sugar is indicated by the liquid's assuming a yellow color, which gradually deepens into a dark brown. If this color does not appear, the non-existence of sugar is certain; but, even when visible, it is always advisable to try *Trommer's* test also. For the details of the somewhat elaborate fermentation-test, and the other tests for sugar, we refer to the text-books upon organic chemistry. *Fehling's* "liquid" furnishes a very sure means of determining the percentage of urea, and also of the sugar, if the quantity of urine passed in twenty-four hours be measured. *Liebermeister* and *Reich*, who, in their many experiments, examined each portion of the urine separately, only noting as reliable those observations which agreed, hardly ever had

occasion to repeat the process in the same portion of urine, after they had conducted their researches for a few days. The polarizing apparatus of *Soleil-Ventzke* is a more convenient and rapid method of ascertaining the percentage of sugar in the urine. The cheaper polarizing apparatus of *Robiquet*, though less accurate, is also applicable to this purpose. In low grades of the malady, the quantity of sugar is not more than from one to two per cent.; in more intense forms, it is as much as six or seven per cent., or more. In well-pronounced instances, the entire amount excreted daily may exceed a pound. The proportion of sugar, however, varies greatly during the progress of the disease, sometimes even fluctuating perceptibly in the course of a day. The causes of these fluctuations are in a great measure unknown, and we are acquainted with a few only of the agents which are capable of increasing or diminishing the percentage of sugar in the urine. Of the former, large draughts of liquid, heavy meals, especially eating large quantities of sugar or of amylaceous matter; of the latter, the exclusion of sugar from the food and drink, particularly the removal of amylaceous matter and similar glycogenous food. The influence of a meal upon the percentage of sugar voided continues for several hours, and then is replaced by a similar discharge of a much more gradual character. Dr. *Moritz Traube* infers, from the great decrease in the elimination of sugar which takes place after several hours' fasting, that, at a certain period during the night, there must be none at all. Had Dr. *Traube* but tested the truth of his conclusion (and he states expressly that he has not done so), had he merely examined a little of this urine of the last few hours of the night, which he supposes should not contain sugar, he would probably find himself to be in the wrong. Among the cases under observation at my clinic, one of which was of quite recent origin, affecting a young, vigorous man, still capable of work, and in the fourteen cases investigated by *Seegen* in Karlsbad, the urine passed in the latter hours of the night and those of early morning invariably contained sugar. Hence Dr. *Traube's* proposed "law" is undeserving of credit until it shall have been proved that cases do occur in which it is as he supposes.

The parching thirst which distresses the patient day and night is easily accounted for, as it has been proved that the polyuria of diabetes is not merely a consequence of much drinking, but rather that the patients drink much because they suffer an excessive loss of water through the kidneys. *Claude Bernard* ascribes the thirst of diabetic patients to the elevated temperature of the liver, which undoubtedly induces an increased absorption in the intestinal canal. This hypothesis, like the others, is idle. A diabetic patient is thirsty on account of the thickening of his blood, much of whose water has been dis-

charged from his kidneys, just as another one thirsts who sweats profusely, or who suffers a great evaporation from his skin from fever, or like a cholera patient who is discharging water through the capillaries of his intestines. A second factor consists in the desiccation of all the tissues as a result of intense endosmotic action from the parenchyma into the vessels. Sugar may be employed as well as salt for the purpose of drying meat in order to preserve it. It is by no means rare for a patient to drink from ten to fifteen quarts of water during the day. The thirst is most intense an hour or two after meals, that is, while the formation of sugar is most active and the production of urine the most copious.

This immense waste of water through the kidneys likewise accounts for the complete arrest of perspiration, and for the decrease in the insensible evaporation from the skin, which have been observed at my clinic. The antagonism between the secretions of the skin and kidneys is as well shown in the dryness of the surface of diabetic people as in the reduction of diuresis, which usually accompanies profuse perspiration. *Griesinger* reports an interesting case, in which sugar was present in the urine and sweat alternately, and in which, whenever the sweat became strongly saccharine, the percentage of sugar in the urine sank one-half. In the aforementioned researches of *Liebermeister* and *Reich*, it was found that a diabetic patient scarcely perspired a third of the quantity given off by a healthy person. Finally, according to the experiments of *Kunde* and *Köhnhorn*, who, by artificial abstraction of water from frogs and mammalia, have induced opacity of the crystalline lenses of these animals, it seems not improbable that the cataract which often accompanies diabetes is a consequence of the excessive loss of water, although indeed the ophthalmologists have raised many objections to this explanation.

An insatiable hunger is a symptom of diabetes, quite as constant as is the unquenchable thirst. It is almost incredible how much food such a patient will consume during a day, often quite regardless of its quality. This voracious hunger, as well as the steadily increasing and finally extreme emaciation, manifestly are due to the fact that a large part of the food eaten is of no profit to the system, because, instead of being devoted to the repair of used-up constituents of the body, it is immediately excreted. Moreover, the consumption of the nitrogenous components of the body is considerably increased in diabetes, as is shown by the augmentation in the quantity of urea produced. Of course there can be no elevation of the bodily temperature, a large part of the heat-producing material passing off from the blood unused.

The impotence, which is nearly always observed in this disease, is probably dependent upon a general decline in the patient's strength

and nutritive condition; nevertheless, the attempt has been made to ascribe diabetic impotence to the drying up of the sperm for want of water, and to a saccharine condition of the semen, supposed to deprive it of its power of inducing nervous excitement.

Finally, we have to mention a series of phenomena, which, although less constant than those heretofore described, still often accompany them and serve to complete the picture of the disease. Caries of the teeth is a symptom which sets in early in the majority of cases. *Falk* explains this phenomenon by supposing that the teeth are exposed to the action of a free acid, formed by decomposition of saccharine secretion within the mouth. Then, again, phymosis and excoriations of the prepuce and glans in men, and of the parts about the meatus urinarius in women, are very distressing occurrences, due probably to wetting of these parts with saccharine urine. Finally, there is often a great tendency to inflammation, ending in necrosis and mortification, exhibited in the very frequent occurrence of furuncles, carbuncles, lobular pneumonia, and pulmonary abscesses and gangrene.

Consumption of the lungs develops as a terminal symptom in many diabetic persons. According to *Griesinger*, nearly one-half of all patients die in this way. Now and then albuminuria is associated with mellituria, thereby augmenting the exhaustion and accelerating the demise of the sufferer. It is not improbable that the parenchymatous nephritis, upon which the albuminuria depends, is a consequence of the constant irritation of the kidney by the presence of sugar in the urine, thus forming a sort of analogue with diabetic balanitis.

The course of diabetes is always chronic, lasting for months and years. In but a very small number of the cases reported, the disease has been acute, terminating fatally within a few weeks, or even sooner. We have scarcely any trustworthy observations of the incipient stages of the malady. Nearly all patients only come under treatment at a period when the profuse urination, tormenting thirst, insatiable hunger, and steadily advancing emaciation, have awakened the suspicion of the existence of serious disease; and they are scarcely ever able accurately to state when the present symptoms first arose, or by what others they had been preceded. Even in the few examples in which the malady has developed quickly, an augmented hunger, thirst, and an unnatural diuresis, were the first manifestations which attracted the attention of the patients and their friends.

Diabetes usually lasts from one to three years. More than sixty per cent. of the cases collected by *Griesinger* terminated fatally within that period. It must, however, be recollected that statistics usually refer to cases treated in hospital, while the majority of those treated in private practice are never published. There is no doubt

that patients in comfortable circumstances, and who possess the means of taking good care of themselves, hold out against diabetes much longer than those who are obliged to seek refuge in hospitals. A complete and permanent recovery from diabetes (if it ever occur at all) is extremely rare; although plenty of cases have been recorded in which a pause in the symptoms, of longer or shorter duration, has been observed. Death, when not the result of some intercurrent complication, usually takes place with the signs of extreme marasmus. Sometimes nervous symptoms arise shortly before death, calling to mind the characteristics of uræmic intoxication.

TREATMENT.—Numerous remedies and "cures" have been recommended for diabetes; unfortunately, most of them have been devised upon principles based on purely hypothetical views as to the nature of the disease, and very few of them rest upon facts established by experience. We shall not discuss procedures such as that whose object is to hinder the conversion of amylum into sugar by exhibition of acids; or that in which nitrogen is to be supplied to the system by administration of ammonia; or in which it is sought to allay irritability of the kidneys by means of opium; or to act upon the liver by means of ox-gall, or gallic acid, or any of the other purely theoretical suggestions. We are indebted to *Griesinger* for a positive experimental demonstration of the inefficacy, and in some cases even the detrimental character, of some of these modes of treatment, such as that by alcohol, rennet, yeast, sugar—intended to supply the place of that lost from the blood.

Experience has also established the fact of the beneficial influence of certain dietetic rules, the first hints of which, however, were derived from theoretical reasoning. It is of the utmost importance that diabetic patients should live principally upon animal food, and that they should eat but very little starchy or saccharine matter. The absolute prohibition of food containing starch and sugar has been abandoned of late; experience having taught, in the first place, that although the loss of sugar might undergo temporary diminution by this procedure, yet it could not cure the diabetes; and besides, it has been found that there are very few patients who for years can endure a diet consisting exclusively of meat, eggs, fish, oysters, crabs, salad, and *Bouchardat's* bran-bread. It relieves the patients greatly, in carrying out the rest of the treatment, to allow them to eat a small portion of bread daily; and it does them no material harm. If we do not allow them this indulgence, we run the risk of their soon becoming so impatient, at the excessive restraint, as to refuse further obedience, and make up for lost time by eating copiously of bread and fruit, for which they almost always have a great desire. Besides the meats, the patient may be allowed such vegetables as do not contain either sugar or starch, or

but very little of them. *Bouchardat* has proved, by the comparison of the articles of food which a diabetic may take without prejudice, that it is quite possible to keep up a sufficient degree of variety in the character of the meals. The list of articles which he permits is about as follows: All sorts of meats roasted, stewed, and even dressed with spices, but not with flour; fresh water fish and marine fish, in eating which the want of bread is less felt than in eating meat; oysters, muscles, crabs, lobsters, etc.; eggs in all the forms known to the culinary art; rich, good cream, but no milk; of the vegetables, spinach, artichokes, asparagus, green beans, the different varieties of cabbage; of the salads, water-cresses, endives, lettuce; of fruits, strawberries and peaches. Although the loss of sugar is augmented by very free supply of liquid, and decreased by a privation of it, yet it is not advisable to forbid the patient to quench his thirst. *Griesinger*, who has experimented upon this subject also, concludes that there is no rapid or considerable decrease in the loss of sugar until the thirsting has been pushed to the point of general disturbance of the system; and, as such a condition can only be endured for a very short time, he allows the patient just enough liquid to slake his thirst. Besides spring-water, acidulated water may be allowed as a beverage, as well as coffee, well-fermented beer, and wine, if not too new, especially red wine.

Inasmuch as diabetic persons are very liable to take cold, and show a predisposition to pneumonia, and the like, directions regarding their clothing must also be laid down, and especially must they be urged to wear flannel next the skin.

Besides these dietetic rules, under the conscientious observation of which diabetes, although incurable, is often tolerated for a great length of time without appreciable decline (as can be proved by many well-attested instances), there are certain medicinal remedies whose effects in diabetes are very decided. In private practice, no credit should be given to the claims of any drug based upon the bare assertion that it has benefited or cured a case of diabetes, particularly when it has been known to fail in other instances. But even those remedies which only exert a slight influence upon the course of the disease (proved, however, by accurate and continuous measurement of the urine, sugar, weight of the body, etc.) should be urgently recommended to private patients by their physicians. At present, however, the alkaline carbonates are the only medicines whose beneficial action upon diabetes can be claimed with certainty. In *Griesinger's* clinic, the exhibition of doses of bicarbonate of soda effected a distinct although slight improvement in the disease. The equally well-established success of the springs of Carlsbad and Vichy have been much

more marked. The old reputation of the Carlsbad waters, as a cure for diabetes, has vindicated itself most brilliantly, according to the observations of *Seegen*. According to him, there can no longer be any doubt that in many cases of diabetes mellitus, a course of several weeks at these springs results in an abatement of the thirst, a decrease in the urination, a gain in weight, *and in a disappearance of the sugar from the patient's urine*. Whether or not these results be permanent, or merely transitory, still, in our present state of knowledge, a course of waters at Carlsbad is the measure which should deserve the chief reliance as a remedy for diabetes mellitus.[3]

CHAPTER VIII.

DIABETES INSIPIDUS.

DIABETES INSIPIDUS and diabetes mellitus, in spite of the agreement of their most conspicuous symptoms, polyuria and unquenchable thirst, are altogether distinct diseases. In the former, the urine does not contain any foreign ingredients, the presence of which might account for the symptoms.

It is true that not long since, in the urine of a patient with diabetes insipidus, *Mosler* discovered inosite, which is not among the normal constituents of urine, and he advanced the hypothesis that inosite played the same part in diabetes insipidus (inosuria) that sugar does in diabetes mellitus (mellituria). But the very slight amount of inosite excreted in the urine of *Mosler's* patient would render this idea improbable, and it is entirely refuted by one of my pupils, Dr. *Strauss*, who wrote an excellent monograph on diabetes insipidus.*

After finding inosite in the urine of two cases of diabetes insipidus—in one case 0.1474 grm. in 6,700 ccm. of urine, in the other 1.508 grm. in 9,600 ccm. of urine—*Strauss* caused three healthy persons, in whose urine there was no inosite, to drink large quantities of water (about ten quarts in twenty-four hours); after this inosite was found in the urine of all three patients, in about the same proportion as in the patients with diabetes insipidus. These beautiful experiments show that a body which normally exists in the kidneys, liver, lungs, and muscles, but, as it undergoes changes in the body, is not found in normal urine, may be excreted through the kidneys by giving large quantities of water.

* Die einfache zuckerlose Harnruhr, von Dr. *F. Strauss*. Tübingen, 1870. Verlag der Lauppschen Buchhandlung.

The most specious of the hypotheses, as to the pathogeny of insipid diabetes, is that which ascribes the polyuria to derangement of innervation of the blood-vessels of the kidney. If the afferent vessels of the Malpighian capsules were to become dilated, in consequence of paralysis of their walls, the pressure within the glomeruli would increase, and with it the rate of filtration of the urine would augment, thus giving rise to polyuria. This primary symptom would be accompanied secondarily by polydipsia. When large quantities of water are withdrawn from the blood, as a consequence of profuse urination, then (just as also occurs after profuse sweating, or intense liquid diarrhœa) the fluids from the interstices of the tissues are greedily absorbed by the now concentrated blood, thus giving rise to an increased need for liquid, and to a sense of severe thirst. Although I regard this as probably a true explanation, and although it acquires further support from the discovery of *Bernard*, that polyuria may be induced in animals by wounding their medulla oblongata at a point somewhat farther up than that at which the so-called "diabetes puncture" is performed, yet I am by no means inclined to regard it as absolutely correct.

With regard to the somewhat obscure etiology of diabetes insipidus, we must confine ourselves to the announcement of the following facts: The disease is more common in males than in females, and in youth than during middle age. I cannot confirm the assertion that old age is exempt from it, as I have seen a case in a patient fifty years old. Its exciting causes have so often been referred to mental affections, to the drinking of cold beverages while the body was overheated, to cold, to over-exertion, to abuse of spirituous liquors, and other agencies, of a nature so various, and of such common occurrence, that the dependence of the malady upon their influence must be regarded as questionable.

It is different with the observations of cases where diabetes insipidus has occurred after injuries of the skull, and in the course of acute and chronic diseases of the brain. These cases, in connection with the above-mentioned experiments of *Bernard*, are peculiarly interesting.

Anatomical Appearances.—The results of the small number of autopsies held upon persons who have suffered from diabetes insipidus agree so imperfectly, that nothing positive can be affirmed regarding the anatomical lesions from which the malady originates. Thus, in a case described by *Neuffer*, the kidneys, contrary to all expectation, were found small and atrophied, while in another, mentioned by *Lebert*, they were enlarged and hypertrophied.

Symptoms and Course.—The amount of urine discharged by a

patient with diabetes insipidus is as great as, or even greater than, that passed in diabetes mellitus. In twenty-four hours it often amounts to from 10,000 to 15,000 ccm. (ten to fifteen quarts), and sometimes even more.

A little girl, ten years old, with diabetes insipidus, who was very undeveloped, and only weighed twenty-three pounds, during her stay in the Tübingen clinic, passed daily an amount of urine that weighed about one-third as much as her body. The urine is very limpid, and, in contradistinction to diabetic urine, of low specific gravity. It rarely rises above 1.005, and often sinks to 1.001 or 1.0005. The relative amount of urea and salts in the urine is low, but the absolute amount of urea excreted in twenty-four hours is usually normal or somewhat increased. In one case that I observed, where the patient consumed a moderate quantity of nitrogenous material, he passed 9,000 ccm. of urine, containing 38 grm. of urea, in twenty-four hours. Rarely the amount of urea passed in the twenty-four hours is diminished. Thus, in one of the cases in my clinic, published by *Strauss*, where the patient weighed about 114 pounds and had good diet, he passed only 23.42 grm. of urea in the twenty-four hours.*

The excretion through the skin and lungs seemed to be considerably reduced, judging by the relative quantities of the liquid drank and urine voided. Doctors *Schmidtlein* and *Spaeth*, who for some time ate and drank exactly the same quantities of food and drink taken by a patient with diabetes insipidus at my clinic, in twenty-four hours excreted from two thousand to twenty-six hundred grammes through their lungs and skin, while the patient only lost from five hundred and forty to six hundred and forty grammes by the same way and in the same time.

Strauss's observations also led to the result that, taking the same amount of fluid, the patients passed from five hundred to two thousand centimetres more urine than healthy persons, while, in the latter, the loss of weight by insensible perspiration was considerably more than in the former.

* In some cases of diabetes insipidus the urine is said to have a very high specific gravity, and to contain an abnormal amount of urea. In this form of disease, about which the observations are very scanty, the original disease would consist of an increased destruction of tissue; the great thirst, as well as the increased secretion of urine, would be very insignificant as compared with the severe constitutional disturbance, and would be explained as in diabetes mellitus. The serum of the blood is abnormally concentrated, and, on endosmotic principles, draws water from the tissues. We shall not further consider this obscure and problematical form of diabetes insipidus.

Strauss justly regards this as another proof that polydipsia is not the cause of polyuria, but the reverse. He writes as follows:

"What reasonable and satisfactory grounds are there for assuming that the cutaneous and pulmonary exhalations of a polydiptic person should differ from those of a healthy person who voluntarily drinks copiously of water?"

But if we suppose a polyuria with consequent concentration of the blood, and decrease of water in the tissue, there would be nothing strange in a diminished gaseous excretion of water by the skin and lungs. In our cases, as in most others, among the symptoms referring to the skin, we find mentioned dryness, harshness, and frequent itching, while profuse perspiration is not mentioned in a single case.

The quantity of liquid imbibed daily, including that contained in the food, is said to have amounted sometimes to sixty or eighty pints. Of course, such a quantity could never permanently be less than that of the urine secreted, without the occurrence of a corresponding decrease of the weight of the body. All cases, wherein it is claimed that the quantity of urine secreted exceeded that of the liquid imbibed, must have been inaccurately observed. !

In my case, the sense of hunger was considerably augmented. *Trousseau* also tells of a patient who ate such enormous quantities that, at a certain restaurant where bread was furnished *à discretion*, they paid him to stay away. In this disease (unlike diabetes mellitus, in which a part of the food consumed, instead of going to repair the used-up constituents of the body, is voided unused), this unnatural hunger—which, moreover, is not a constant occurrence—can only be accounted for by supposing that the exaggerated consumption and discharge of water accelerate the waste of the nitrogenous elements of the body. It may be accepted as a law that, during an accelerated flow of the juices of the parenchyma through the organs, a larger quantity of albuminous material is consumed.

In some patients the general health and vigor long remain undisturbed. A girl twenty years of age, with insipid diabetes, who remained under observation at my clinic for a considerable time, retained her blooming aspect, and could even do hard work without special effort. In other patients, signs of digestive derangement, cardialgia, vomiting, irregularity of the bowels, emaciation, and an unaccountable sense of debility, set in early. In the instance reported by *Neuffer*, the patient died with these symptoms, no appreciable cause of death being discoverable upon autopsy. The course and duration of the disease vary. In some cases it develops gradually, in others it sets in suddenly. Not unfrequently a transient improvement in the symptoms is observed. It may also happen that, during the attack

of some intercurrent disease, the daily flow of urine becomes normal, and returns to its former profusion after the attack has terminated. As a rule, the malady lasts for years without imperilling life. It is only in rare instances, as in that of *Neuffer*, already alluded to, that death takes place without the intervention of some other disease. Complete and permanent recovery from diabetes insipidus is likewise an extremely rare event.

Treatment.—It is always suspicious when numerous remedies are recommended for a disease; in such cases, usually, none of them have any special effect on the course and termination of the affection. This is also true of the recommendations of saltpetre, in the shape of sal prunellæ, of valerian, belladonna, opium, ergotine, creosote, and other remedies in diabetes insipidus. They are not based on the results of experience, but on theoretical grounds. I have not used any of these remedies persistently and energetically, as my patients bore their disease pretty well—so that there was no necessity for energetic treatment. On the whole, I would advise you, in treating simple diabetes, to limit yourselves to preventing the bad results induced by the disease, or to combating them if they have already appeared. Especial attention should be paid to the disturbances of digestion and nutrition, which are not uncommon. Rational diet, combined with cod-liver oil, malt extract, and iron, improved the condition of the little girl above-mentioned, without diminishing her thirst, or the secretion of urine; and, although her early death had been expected, she was discharged from the clinic considerably increased in weight and much improved in health.

ADDITIONS TO THE REVISED EDITION OF 1880.

SECTION III.—GENERAL DISORDERS OF NUTRITION.

1.—P. 803.

According to *Virchow*, in obstinate cases of chlorosis there is defective development and small size of the aorta and vessels generally. The aorta may be so small as not to admit the little finger, while its walls may be uncommonly thin; they, however, retain their elasticity, unless there has been fatty degeneration of the inner and middle coats. This failure of vascular development is sometimes accompanied by stunted growth of the whole body. The heart is sometimes small, but is usually normal, or slightly dilated; exceptionally it is hypertrophied. There is an inclination

to endocarditis of the left ventricle. In women with this trouble of the heart and blood-vessels, child-bearing may induce ulcerous endocarditis. Feverish diseases are more dangerous in chlorotic persons than in others. In giving iron in chlorosis, it is well to administer it after eating. Mineral waters containing pyrophosphate of iron may be drunk at meal-times. This drug may have to be discontinued when there is gastric disturbance or fever. Iron is to be aided by good, easily digestible diet, and residence in a healthy locality. In chlorosis and anæmia generally, alcoholic beverages may be useful by impeding transformation of tissue, but should be avoided if they induce excitement, palpitation, rush of blood to the head, etc. Trial must decide whether wine or beer answers the best purpose.

2.—P. 832.

Of late it has been considered possible that the use of milk from cows with murrain might have an injurious effect, because formations similar to those in tubercles in men have been found in the anthrax-nodules of cattle, and because experiments have shown that feeding other animals on the milk of these cows will give them tuberculosis and diseases of the lymphatic glands of the mesentery and neck, similar to those in scrofula. Hence the use of such milk may be suspected as a cause of the trouble, but has not yet been proved to be so.

In the treatment of scrofula considerable reputation is enjoyed by the mud-baths of Nauheim, Kreuznach, etc., in Germany; they increase the transformation of tissue, and prove advantageous for the glandular enlargements, as well as for the affections of the mucous membrane and skin. When cod-liver oil is given, it is sometimes better borne when not taken on an empty stomach, but half an hour or so after eating. Pale, anæmic children often require some preparation of iron. When the patient is not anæmic or feeble, at proper seasons it is often well to order a laxative mineral water, such as a continued moderate use of Carlsbad water. The waters containing iodine have so very small a percentage of it, that it is doubtful if it has any special effect.

3.—P. 843.

There have been numerous disputes as to the glycogene builders; among them are grape, cane, milk, and fruit sugar, inulin, glycerin, etc. Amylaceous food has the greatest effect on the formation of sugar; under its use the portal vein is always rich in dextrin. It

is not known just how sugar is made from the food; it normally exists in the blood in such slight quantity that it does not transude into the urine; in the blood of diabetic patients *Cantani* found it as high as 8 parts in 1,000.

Artificial mellituria may be induced by dividing the inferior cervical ganglia, or by irritating the floor of the fourth cerebral ventricle (which at the same time induces polyuria); division of the splanchnics has no effect. *Schiff* induced diabetes by destruction of the spinal medulla before and behind the origin of the brachial nerves, etc. Artificial diabetes may also be induced by injections of curare, solutions of common salts, some of the salts of soda, nitrite of amyl, morphine, etc.; this effect is prevented by previous division of the splanchnics.

Diabetes is a disease of the vasomotor nerve-system; on autopsy the liver is found normal, but there are frequently changes in the brain (tumors, etc.). Possibly the sugar formed during digestion of amylaceous substances, being taken up by the lymphatics and portal vessels, enters the blood directly. *Cantani* regards sugar in the blood as necessary to life, and thinks it is one source of strength; it appears as grape-sugar, and is broken up by some ferment; if the organs furnishing this ferment (the pancreas?) be diseased, the sugar appears in the urine. In the first stages of diabetes, by withdrawing amylaceous food, the collection of sugar in the blood and its excretion by the urine may be prevented, and by continuing this treatment for some time the disease may perhaps be entirely cured; even later in the disease, the excretion of sugar may be arrested, but it is doubtful if the disease can be cured. Although the above theories are not universally accepted, the treatment advocated is probably the most successful of the many that have been tried.

Possibly diabetes may depend on a variety of causes, having as a common result the non-consumption of sugar and its excretion by the urine. *Senator* divides it into neurogenous, gastro-enterogenous, and hepatogenous forms. The first would include the cases caused by injuries, tumors, etc., affecting the nervous system; the second, the *Cantani* cases; and the third class, cases where, as a result of hyperæmia of the liver, hepatic sugar passes more rapidly into the blood. There would seem to be either two forms of diabetes, or else two stages of the same disease, in one of which exclusion of amylaceous substances from the diet prevents the appearance of sugar in the urine.

Diabetic urine often has a peculiar odor, which is ascribed to aceton. *Böttcher* tests for sugar by adding to urine in a test-tube

a small quantity of nitrate of bismuth ; then freely adding a concentrated solution of carbonate of soda, or some potash lye ; then heating to the boiling-point. If sugar be present, the bismuth turns gray, then black. This test may be used in addition to others, but is less delicate than the copper test.

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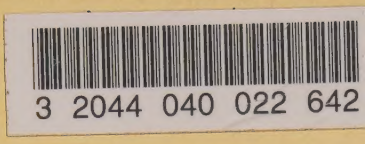
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